Mucocutaneous Presence of Cytomegalovirus Associated With Human Immunodeficiency Virus Infection

Discussion Regarding Its Pathogenetic Role

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Objectives: To investigate the significance of cytomegalovirus (CMV) in mucocutaneous lesions in patients with human immunodeficiency virus (HIV), and to elucidate its pathogenetic role in lesions genesis.

Design: Retrospective (study 1) and prospective (studies 2 and 3) surveys.

Setting: Departments of Dermatology, Pathology, and Microbiology at a university hospital in Madrid, Spain.

Patients: Seventeen HIV-infected patients with CMV presenting any type of mucocutaneous lesions (study 1); 27 HIV-positive patients with mucocutaneous vesicles and/or ulcers of any type and location (study 2); and 12 severely immunosuppressed HIV-positive volunteers (study 3).

Interventions: Mucocutaneous biopsy specimens from the lesions (studies 1 and 2) and from nonlesional skin (study 3) were analyzed by light microscopy, immunohistochemical analysis, and microbiological analysis (standard viral culture and shell-vial technique).

Main Outcome Measures: Clinical data; histologic, immunohistochemical, and microbiological findings.

Results: (1) Studies 1 and 2: Most of the lesions where CMV was found were ulcers localized mainly on perianal, genital, and perigenital areas, usually as part of polymicrobial infections, particularly herpes simplex and varicella-zoster virus infections. The finding of CMV was confirmed in all cases by light microscopy; microbiological analysis was rarely useful. The finding of mucocutaneous CMV inclusions allowed their early detection in extracutaneous locations. (2) Study 3: Cytomegalovirus was present on healthy skin of the perianal area in 3 patients, and on the forearm in 1 patient.

Conclusion: Cytomegalovirus does not play any significant pathogenetic role at least in most of the cutaneous lesions where it is found.

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The cytomegalovirus (CMV) is a β-herpesvirus that has recently become important as an opportunistic agent in different immunosuppressive conditions, particularly in human immunodeficiency virus (HIV) infection. There are still numerous controversial aspects of the natural history of CMV infection and its pathogenetic role in the induction of lesions in the tissues, especially in the skin and in the mucous membranes. To better characterize the significance of CMV in mucocutaneous lesions in HIV-infected patients, and to elucidate its pathogenetic role, we performed a clinicopathologic and microbiological study with 3 steps.

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PATIENTS AND METHODS

STUDY 1

From March 1987 until December 1999, all biopsy material and records of 17 patients infected with the HIV with CMV in any type of mucocutaneous lesions were retrospectively reviewed. We performed immunohistochemical analysis on formalin-fixed, paraffin-embedded tissue. We applied a standard protocol with the avidin-biotin-peroxidase method, using monoclonal antibodies against the immediate early and early antigens of human CMV (1:25 Dako; Copenhagen, Denmark). The biopsy specimens and the exudate of the lesions were cultured and incubated for at least 1 month in a monolayer of human embryonic pulmonary fibroblasts (MRC-5 cell line) using standard viral culture. For the shell-vial technique, we used fluorescent antibodies against immediate early and early antigens of human CMV (CMV Microtrak; Syva Company, St Louis, Mo), with review after 24 and 48 hours.

STUDY 2

Based on study 1, which demonstrated that the mucocutaneous lesions in which CMV was most frequently found were vesicles or ulcers, we started a prospective study including 27 consecutive patients infected with HIV with mucocutaneous vesicles and/or ulcers of any type and location. A lesional skin or mucous membrane biopsy specimen was analyzed, both histologically (multiple sections stained with hematoxylin-eosin) and microbiologically (standard viral culture and shell-vial technique). The exudates from the ulcers or the content of the vesicles were also cultured microbiologically. Informed consent was obtained from all patients.

STUDY 3

Based on studies 1 and 2, in which the presence of CMV was most prevalent in the periana1 area, and considering the hematological dissemination of the CMV, we carried out a prospective study on 12 patients who came to our department with different dermatoses. All the patients were infected with HIV and were severely immunosuppressed (CD4+ cell count less than 150 cells/µL). We performed 2 nonlesional cutaneous biopsies on the periana1 region and the external aspect of the forearm, respectively. They were studied histologically and microbiologically. Informed consent was obtained from all patients.

We studied 27 HIV-infected patients, 4 women and 23 men (aged 23-54 years). Most were either homosexuals (13/27) or injection drug users (10/27). All but 4 (85.2%) had a CD4+ cell count lower than 200 cells/µL. Antibodies against CMV were found in all patients but 1. The CMV was isolated from neither the biopsy specimens nor from ulcer exudates. The shell-vial technique detected CMV in the skin of only 1 patient.

All patients had antibodies against CMV (titers ranging from 1:8 to 1:2048) and were severely immunosuppressed (fewer than 100 CD4+ cells/µL in 15 of 17 patients). In 4 patients, the demonstration of CMV was carried out simultaneously with the diagnosis of HIV infection. The finding of cytomegalic cells in mucocutaneous lesions led to the simultaneous detection of CMV in other tissues and/or fluids in 6 patients. During the follow-up, the early detection of CMV in 5 more patients resulted in a prompt treatment. The extracutaneous involvement included the retina, central nervous system, lung, esophagus, stomach, colon, rectum, liver, and blood. Most of the cutaneous lesions disappeared when treated with acyclovir, even in 3 cases in which neither HSV nor VZV were detected. New outbreaks had a satisfactory response with the same treatment. The patient with bacillary angiomatosis did not respond to ganciclovir but was cured with erythromycin. The follow-up ranged from 1 to 36 months and, at the end of it, 11 patients had died and 5 were in a terminal situation. The remaining patient was lost to follow-up.
luetic oral ulcers, and 2 patients with nonetiologically diagnosed ulcers. Only 2 patients had received acyclovir prior to the study. Findings of a histopathologic evaluation revealed CMV in 6 patients (22.2%), all of whom had perianal ulcers. In 4 patients, CMV coincided with signs of herpetic infection in the epidermis from the same sections; in 1 patient, HSV was cultured from the same lesion. Cytomegalovirus was found alone in the remaining patient, who had received treatment with acyclovir during the previous 7 days. Five patients were homosexual and 1 was an injection drug user. They were all severely immunosuppressed (CD4 cell count lower than 100 cells/µL). The CMV was isolated (using the shell-vial technique to analyze the biopsy specimens) in only 1 patient. The standard viral culture (biopsy specimen and exudate) and shell-vial technique of the exudate demonstrated the presence of HSV-1, HSV-2, and VZV, but not of CMV. The detection of CMV in the skin allowed a subsequent finding of CMV in different extracutaneous locations (meninges, retina, lung, and blood) in 4 patients.

**STUDY 3**

We studied 12 patients (11 men and 1 woman) aged from 26 to 54 years. Nine of them were homosexuals, 1 was heterosexual, and 1 was an IDU. All of them had antibodies against CMV. The presenting problems varied: 9

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**Table 1. Clinical Data Regarding the Patients With CMV in Mucocutaneous Lesions (Study 1)**

<table>
<thead>
<tr>
<th>Patient No./Sex/Age, y</th>
<th>Risk Factor</th>
<th>Mucocutaneous Lesions</th>
<th>Etiological Diagnosis</th>
<th>Duration, d</th>
<th>With Acyclovir†</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/F/24</td>
<td>IDU</td>
<td>Crusted vesicles (trunk, extremities)</td>
<td>Disseminated herpes zoster</td>
<td>10</td>
<td>No</td>
</tr>
<tr>
<td>2/M/30</td>
<td>Homosexual</td>
<td>Ulcers (perianal and scrotal)</td>
<td>Unknown</td>
<td>30</td>
<td>Yes</td>
</tr>
<tr>
<td>3/M/39</td>
<td>Homosexual</td>
<td>Ulcerated nodule (maleolar)</td>
<td>Bacillary angiomatosis</td>
<td>120</td>
<td>No</td>
</tr>
<tr>
<td>4/F/45</td>
<td>Heterosexual (promiscuous)</td>
<td>Crusted vesicles (face, trunk, and legs)</td>
<td>Disseminated herpes zoster</td>
<td>21</td>
<td>No</td>
</tr>
<tr>
<td>5/M/44</td>
<td>Homosexual</td>
<td>Ulcer (perianal)</td>
<td>Unknown</td>
<td>30</td>
<td>No</td>
</tr>
<tr>
<td>6/M/35</td>
<td>IDU</td>
<td>Ulcers (oral)</td>
<td>Unknown</td>
<td>7</td>
<td>No</td>
</tr>
<tr>
<td>7/M/35</td>
<td>Homosexual</td>
<td>Ulcers (perianal)</td>
<td>Herpes simplex</td>
<td>45</td>
<td>No</td>
</tr>
<tr>
<td>8/M/54</td>
<td>Homosexual</td>
<td>Ulcers (scrotal)</td>
<td>Herpes simplex</td>
<td>60</td>
<td>Yes</td>
</tr>
<tr>
<td>9/F/56</td>
<td>Heterosexual (promiscuous)</td>
<td>Ulcers (oral)</td>
<td>Herpes simplex</td>
<td>3</td>
<td>No</td>
</tr>
<tr>
<td>10/M/31</td>
<td>Homosexual</td>
<td>Ulcers (perianal and perigenital)</td>
<td>Herpes simplex</td>
<td>60</td>
<td>Yes</td>
</tr>
<tr>
<td>11/M/31</td>
<td>IDU</td>
<td>Ulcers (perianal)</td>
<td>Herpes simplex</td>
<td>10</td>
<td>No</td>
</tr>
<tr>
<td>12/M/33</td>
<td>Homosexual</td>
<td>Ulcer (perianal)</td>
<td>Herpes simplex</td>
<td>30</td>
<td>Yes</td>
</tr>
<tr>
<td>13/M/39</td>
<td>Homosexual</td>
<td>Ulcers (perianal)</td>
<td>Herpes simplex</td>
<td>10</td>
<td>No</td>
</tr>
<tr>
<td>14/M/38</td>
<td>Homosexual</td>
<td>Ulcers (perianal)</td>
<td>Herpes simplex</td>
<td>45</td>
<td>No</td>
</tr>
<tr>
<td>15/M/53</td>
<td>Homosexual</td>
<td>Ulcers (perianal)</td>
<td>Herpes simplex</td>
<td>90</td>
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<tr>
<td>16/M/37</td>
<td>Homosexual</td>
<td>Ulcers (perianal)</td>
<td>Unknown</td>
<td>30</td>
<td>Yes</td>
</tr>
<tr>
<td>17/F/34</td>
<td>IDU</td>
<td>Ulcers (oral and perioral)</td>
<td>Herpes simplex</td>
<td>120</td>
<td>No</td>
</tr>
</tbody>
</table>

* CMV indicates cytomegalovirus; IDU, injection drug user.
† Treatment with acyclovir within 1 month before the histologic and microbiological studies of the lesions were performed.

**Table 2. Histologic and Microbiological Data Regarding the Patients With CMV in Mucocutaneous Lesions (Study 1)**

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Light Microscopy</th>
<th>IHCh</th>
<th>Biopsy Specimen</th>
<th>Exudate</th>
<th>Standard Viral Culture</th>
<th>Shell-Vial Technique</th>
<th>Standard Viral Culture</th>
<th>Shell-Vial Technique</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>CMV, HI, ESS</td>
<td>CMV</td>
<td>NP</td>
<td>NP</td>
<td>NP</td>
<td>NP</td>
<td>NP</td>
<td>NP</td>
</tr>
<tr>
<td>2</td>
<td>CMV</td>
<td>CMV</td>
<td>NP</td>
<td>NP</td>
<td>NP</td>
<td>NP</td>
<td>NP</td>
<td>NP</td>
</tr>
<tr>
<td>3</td>
<td>CMV, BA</td>
<td>CMV</td>
<td>Neg</td>
<td>NP</td>
<td>Neg</td>
<td>NP</td>
<td>NP</td>
<td>NP</td>
</tr>
<tr>
<td>4</td>
<td>CMV, HI</td>
<td>CMV</td>
<td>Neg</td>
<td>NP</td>
<td>Neg</td>
<td>NP</td>
<td>NP</td>
<td>NP</td>
</tr>
<tr>
<td>5</td>
<td>CMV</td>
<td>CMV</td>
<td>Neg</td>
<td>NP</td>
<td>NP</td>
<td>NP</td>
<td>NP</td>
<td>NP</td>
</tr>
<tr>
<td>6</td>
<td>CMV</td>
<td>CMV</td>
<td>NP</td>
<td>NP</td>
<td>NP</td>
<td>NP</td>
<td>NP</td>
<td>NP</td>
</tr>
<tr>
<td>7</td>
<td>CMV, HI, ESS</td>
<td>CMV</td>
<td>HSV-2</td>
<td>NP</td>
<td>HSV-2</td>
<td>NP</td>
<td>HSV-2</td>
<td>NP</td>
</tr>
<tr>
<td>8</td>
<td>CMV</td>
<td>CMV-1</td>
<td>HSV-2</td>
<td>NP</td>
<td>HSV-2</td>
<td>HSV-2</td>
<td>HSV-2</td>
<td>HSV-2</td>
</tr>
<tr>
<td>9</td>
<td>CMV</td>
<td>CMV</td>
<td>HSV-1</td>
<td>HSV-1</td>
<td>HSV-1</td>
<td>HSV-1</td>
<td>HSV-1</td>
<td>HSV-1</td>
</tr>
<tr>
<td>10</td>
<td>CMV</td>
<td>HI</td>
<td>HSV-2</td>
<td>HSV-2</td>
<td>HSV-2</td>
<td>HSV-2</td>
<td>HSV-2</td>
<td>HSV-2</td>
</tr>
<tr>
<td>11</td>
<td>CMV</td>
<td>HI</td>
<td>HSV-2</td>
<td>HSV-2</td>
<td>HSV-2</td>
<td>HSV-2</td>
<td>HSV-2</td>
<td>HSV-2</td>
</tr>
<tr>
<td>12</td>
<td>CMV</td>
<td>HI</td>
<td>Neg</td>
<td>HSV-2</td>
<td>HSV-2</td>
<td>HSV-2</td>
<td>HSV-2</td>
<td>HSV-2</td>
</tr>
<tr>
<td>13</td>
<td>CMV</td>
<td>HI</td>
<td>HSV-2</td>
<td>Neg</td>
<td>HSV-2</td>
<td>HSV-2</td>
<td>HSV-2</td>
<td>HSV-2</td>
</tr>
<tr>
<td>14</td>
<td>CMV</td>
<td>HI</td>
<td>HSV-2</td>
<td>HSV-2</td>
<td>HSV-2</td>
<td>HSV-2</td>
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<tr>
<td>15</td>
<td>CMV</td>
<td>HI</td>
<td>HSV-2</td>
<td>HSV-2</td>
<td>HSV-2</td>
<td>HSV-2</td>
<td>HSV-2</td>
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</tr>
<tr>
<td>16</td>
<td>CMV</td>
<td>HI</td>
<td>HSV-2</td>
<td>HSV-2</td>
<td>HSV-2</td>
<td>HSV-2</td>
<td>HSV-2</td>
<td>HSV-2</td>
</tr>
<tr>
<td>17</td>
<td>CMV</td>
<td>HI</td>
<td>HSV-1</td>
<td>HSV-1</td>
<td>HSV-1</td>
<td>HSV-1</td>
<td>HSV-1</td>
<td>HSV-1</td>
</tr>
</tbody>
</table>

* CMV indicates cytomegalovirus; IHCh, immunohistochemical analysis; HI, herpetic infection; ESS, eccrine squamous syringometaplasia; NP, not performed; BA, bacillary angiomatosis; Neg, negative; HSV, herpes simplex virus.
patients had perianal ulcers (7 with HSV infection [3 of whom also had cytomegalic inclusions] and 2 with CMV alone); 1 had lumbar-sacral herpes zoster; 1, perianal condylomas; and 1, herpetic oral ulcers. A histopathologic study on healthy skin of the perianal area revealed the presence of CMV in 3 cases. In 1 case it was found in the forearm. The latter 3 patients had surrounding perianal ulcers with CMV. Standard viral culture and shell-vial findings were negative for CMV in all cases. The CMV was detected in extracutaneous locations in 4 patients (including the 3 with CMV in healthy skin).

Cytomegalovirus infection is common in HIV-infected patients, but its presence in mucocutaneous lesions is rarely reported. The reported clinical presentations have been very variable, including ulcers, vesicles, purpuric macules, verrucous lesions, prurigo nodularis–like lesions, erythematous and crusted papules, and digital infarcts.3–12 Several studies with variable and even contradictory results have investigated the role of CMV in ulcers in different locations in HIV-positive patients.13–19 Our data confirm that while there were no characteristic mucocutaneous lesions diagnostic for CMV infection, the presence of persistent anogenital ulcers and, less frequently, oral ulcers in HIV-infected patients should make one suspect and search carefully for the presence of CMV in these lesions.

There is a controversy regarding the comparative sensitivity among the different diagnostic techniques used to determine the presence of CMV in mucocutaneous lesions.18,20,21 From our experience, light microscopy with hematoxylin-eosin stain is the most sensitive tool. Because the number of cells containing CMV is very variable, and sometimes only 1 or 2 infected cells are found in each section of the tissue, we recommend a meticulous search and a serial sectioning of the samples. Cytomegalovirus inclusions are mostly found in the endothelial cells but, occasionally, they have also occurred inside the keratinocytes,5,7 in sweat gland epithelial cells,5 and in macrophages22–24 and fibroblasts.7,25,26 The smears from the ulcers are rarely if ever useful. Immunohistochemical analysis is useful to confirm the diagnosis in certain cases (eg, intense inflammation). The standard viral culture of mucocutaneous biopsy specimens is not useful to diagnose CMV infection. Since coinfection with HSV/VZV is often present, these viruses destroy the monolayer of human fibroblasts and prevent the growth of CMV. The shell-vial technique, with an early evaluation at 24 hours, is an attempt to avoid this problem, but in our series its usefulness was very low.

Nevertheless, the finding of CMV in the skin/mucosa raises several questions: What is the predictive significance of its presence in immunosuppressed patients? Second, is CMV alone capable of inducing tissue damage of the skin and/or mucosa? And finally, why are most CMV lesions located in the perianal region? The interpretation of the presence of CMV in the skin and/or mucosa is controversial. Its presence could indicate that
CMV plays a pathogenetic role, both in the origin of the lesions and in their chronicity. But, it could also simply be the expression of the endothelial colonization that occurs during the course of a hematogenous disseminated infection (sometimes the detection of CMV within the skin is simultaneous with the viremia\(^3\)). It might also be that the CMV has reactivated within the endothelial cells, which have thus acted as reservoirs during the latency period. Or the presence of CMV might be a consequence of an autoinoculation by the shedding of CMV through the feces, the urine, or the saliva. It is difficult to establish a cause-effect relationship between the clinical lesion and the presence of CMV in that lesion. In other words, the presence of CMV does not necessarily indicate the etiology of the pathologic process.

The following could be arguments in favor of a pathogenetic role: (1) The causality of CMV in retinitis has been accepted. However, the same is not true for CMV in the blood, the urine, the lung, or in stool. (2) There are reports of histopathologic reactions (inflammatory infiltrate, necrosis, etc) in association with the presence of CMV and signs of cytopathic viral effect without evidence of other antimicrobial agents.\(^1\) Our experience does not agree with these previous reports. (3) It has been reported that there is a correlation between the intensity of the inflammatory infiltrate and the number of cytomegalic cells that are apparently responsible for that reaction. However, it is also true that the greater number of vessels and inflammatory cells provide increased potential expression of CMV during viremia. (4) There is an absence of CMV in most lesional skin biopsy specimens from different cutaneous diseases in patients with systemic CMV infection and viremia.

Arguments against a pathogenetic role of CMV are as follows: (1) Cytomegalovirus is frequently observed or cultured with other infectious agents that have a recognized pathogenetic role and alone could induce the etiologic process. Apart from the well-known association of CMV with HSV,\(^2,7,14,18,27-30\) its presence has also been associated with Staphylococcus aureus and acid-fast bacilli.\(^22,27,31\) Our results indicate that most mucocutaneous CMV lesions are associated with either HSZ or VZV infection. The reasons for not finding these viruses in some cases could be (a) the minimal portion or absence of epidermis in some biopsy specimens (cases 5 and 6 in study 1); (b) marked epidermal necrosis; (c) false-negative findings in diagnostic techniques\(^6,28,32\); or (d) previous treatment with acyclovir, which might have made the HSV/VZV, but not the CMV, disappear (cases 2 and 16 in study 1). (2) Unexpected findings of CMV have occurred in unrelated skin lesions (patient 3),\(^3\) in 2 other patients with bacillary angiomatosis,\(^3,13,14\) in a case of Kasabian sarcoma,\(^33-36\) and even in a case of a posttraumatic scar,\(^39\) all of which are conditions with a well-developed vascularity. (3) Cytomegalovirus has occurred in apparently healthy skin. In our study 3, 3 patients had CMV in perianal healthy skin, 1 with inclusions on the skin of the forearm, far from the lesions in which CMV had been observed. This finding has also been described in a patient with active extracutaneous CMV infection.\(^39\) And (4) spontaneous healing has occurred of lesions in which CMV was isolated.\(^11,24\)

On the basis of all these data, we believe that CMV does not play any significant pathogenetic role in at least most mucocutaneous lesions where it is found. Whether it contributes to the maintenance of the lesion once the primary pathogen is eliminated cannot be answered. The predilection of CMV for anogenital ulcers has been clearly confirmed in our study. We believe that it is very probable that the cytomegalic inclusions reach the area as a consequence of the autoinoculation after shedding through the feces, urine, or even the semen in a herpes simplex-induced ulcer. It is well known that the colon, an organ with a special tropism for the virus, is frequently colonized.\(^30,31\) The possibility of CMV hematogeneously reaching the granulation tissue of anogenital ulcers or of a reinfeciton through anal coitus in homosexuals cannot be ruled out.

As our study shows, it is very important to search for and detect CMV because its presence frequently represents the first sign of CMV infection,\(^28\) or even the first expression of HIV infection.\(^42\) But apart from this, the detection of CMV has a high prognostic value because its presence in the skin and/or mucosa usually indicates a concomitant generalized CMV infection. This fact should encourage us to search for infection in other tissues (retina, blood, etc) and, if it is found, to start treatment as soon as possible. In summary, on the basis of our findings that CMV is usually associated with HSZ and VZV infections, and is found unexpectedly in cutaneous abnormalities of known diagnosis (ie, bacillary angiomatosis) or even in apparently healthy skin, we believe that the CMV does not play any significant pathogenetic role in at least most cutaneous lesions in which it is found.

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Preliminary results were presented as a poster at the 53rd Annual Meeting of the American Academy of Dermatology, New Orleans, La, February 1995.

The present study was presented as a poster at the Ninth Congress of the European Academy of Dermatology and Venereology, Geneva, Switzerland, October 2000.

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