Cytomegalovirus as a Primary Pulmonary Pathogen in AIDS*

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In patients with AIDS, isolation of cytomegalovirus (CMV) from respiratory secretions is common. It is often found with other pathogens, which has led to debate regarding its role as a primary pulmonary pathogen. A retrospective investigation of patients with AIDS and CMV as a sole pulmonary isolate was performed in an attempt to describe their clinical presentation and course. All patients admitted to the hospital with pneumonia and with BAL or transbronchial biopsy (TBB) specimen positive for CMV between 1991 and 1994 were identified through a review of inpatient records. Inclusion criteria included positive CMV cultures from BAL, cytomegalic inclusion bodies from BAL or TBB, and thorough documentation of the absence of other pulmonary pathogens. Nine patients met the inclusion criteria for CMV pneumonitis. Seven were male and two were female, ages 26 to 44 years, and all had a history of opportunistic infections. Typical clinical presentation was characterized by increased respiratory rate, hypoxemia, and diffuse interstitial infiltrates. The mean CD4 count was 29.6 (±22) cells per cubic millimeter, mean lactate dehydrogenase level was 414 (±301) IU/L, and in seven patients in whom CMV antigen was measured it was greater than 50 positive cells per 200,000 WBCs. Three untreated patients died of respiratory failure and three had autopsy confirmation of CMV pneumonia. Five patients were treated with anti-CMV therapy for at least 2 weeks, and all demonstrated improvement in symptoms, oxygen saturation, and chest radiograph. At 3 months follow-up, all five patients were asymptomatic with no pulmonary symptoms. At 6 months follow-up, three of the five patients remained asymptomatic; the other two died of other opportunistic infections. In at least these nine patients, CMV represented a primary pulmonary pathogen. Patients who were treated responded quickly and were able to be discharged home from the hospital with marked improvement in their symptoms. We recommend that clinicians consider this diagnosis in the proper setting and consider treatment with anti-CMV therapy. (CHEST 1997; 111:128-34)

Key words: acquired immunodeficiency syndrome; cytomegalovirus; diagnosis; pneumonitis

Abbreviations: AFB=acid-fast bacteria; CMV=cytomegalovirus; LDH=lactate dehydrogenase; PCP=Pneumocystis carinii pneumonia; TBB=transbronchial biopsy

H uman cytomegalovirus (CMV), a member of the Herpesviridae family, commonly infects healthy individuals. Rarely, it causes symptomatic disease in immunocompetent adults, although the course is typically benign. Like all herpes viruses, it remains latent for the life of the host.1 Under conditions of immune compromise and impaired cell-mediated immunity, such as AIDS, CMV can reactivate and lead to chorioretinitis, esophagitis, colitis, adrenalitis, and encephalitis.2,3 Although frequently isolated from respiratory secretions of HIV-infected patients,2,4,5 the role of CMV as a primary pulmonary pathogen is uncertain.6,7 CMV has been recorded in 37 to 53% of respiratory specimens from patients with Pneumocystis carinii pneumonia9 (PCP) and in 19 to 43% of patients undergoing bronchoscopy for diagnosis of opportunistic infections.2,4 We describe our experience with nine patients admitted to Yale New Haven Hospital with presumed PCP pneumonia, who ultimately had CMV documented as the sole pulmonary pathogen.

Materials and Methods

Clinical records of all patients with HIV infection admitted to the medical service in Yale New Haven Hospital between
December 1991 and December 1994 were reviewed and those admitted with the diagnosis of pneumonia, pneumonitis, bronchitis, or PCP were identified. Hospital charts were comprehensively reviewed for all patients who underwent bronchoscopy.

Results of all specimens obtained by either BAL or transbronchial biopsy (TBB) were submitted to the virology and microbiology laboratories at Yale New Haven Hospital. Bacteriologic investigation was performed on BAL fluid and TBB specimens and included Gram stains, Ziehl-Neelsen staining, and culture for bacteria, mycobacterium, viruses, and fungi. All specimens underwent histologic and cytologic examination. Hematoxylin-eosin stains, Grocott’s silver stain, and Giemsa stains were performed. Patients who had positive CMV cultures from both BAL fluid and TBB specimens were identified.

CMV Culture Technique

Specimens obtained by BAL were transported in a sterile specimen vial. Three milliliters of well-mixed BAL fluid was mixed with 0.3 mL of stock antibiotic solution containing 0.2 mL vancomycin hydrochloride (50 mg/mL), 1.0 mL gentamicin sulfate (50 mg/mL), and 4.0 mL amphotericin B (250 μg/mL) in 4.8 mL phosphate-buffered saline solution; 0.3 mL of the mixture was then inoculated into two shell vials both containing the MRC-5 continuous cell line and three separate 0.2-mL roller tubes containing MRC-5, A549, and rhMK continuous cell lines, respectively. Tissue from TBB was minced in 3 to 5 mL trypsin/EDTA. The tissue suspension was transferred to 15-mL centrifuge tubes. The cells were pelleted at 2,000 rpm for 10 min and then were inoculated into continuous cell lines as above. Cultures were observed for 3 weeks. Positive cultures were recognized by typical cytopathic effect and confirmed by reaction with fluorescein-conjugated monoclonal CMV-specific antibodies (Dupont NEN; Boston).

CMV Antigen Technique

The CMV antigen level was based on immunocytochemical detection of CMV immediate early antigens in blood leukocytes as previously described.9,10

Case Definition of CMV Pneumonitis

CMV pneumonitis was defined by the universal presence of the following three criteria: (1) positive CMV cultures from both BAL and TBB specimens; (2) cytologic examination that documented the typical cytopathologic condition of CMV—characteristic enlarged cells with large pleomorphic nuclei and intranuclear and cytoplasmic inclusions; and (3) absence of any other pulmonary pathogen identified by bacterial, fungal, viral, or acid-fast stains or cultures.

Medical records of patients identified as meeting criteria for CMV pneumonitis were comprehensively reviewed. We recorded the following specific data: (1) age; (2) gender; (3) race; (4) suspected mode of transmission of HIV; (5) history of opportunistic infections; (6) history of pulmonary infections; (7) signs and symptoms on presentation, including cough, shortness of breath, and dyspnea on exertion; (8) chest radiograph on hospital admission and discharge; (9) serum WBC count, electrolytes, lactate dehydrogenase (LDH), CD4; (10) PaO2 on admission and arterial oxygen saturation (SaO2) prior to hospital discharge; (11) CMV antigen and CMV culture; (12) all bacterial, mycobacterial, and fungal smears and cultures from any other tissue source; (13) therapeutic interventions, including antibacterial, antiviral, and steroid therapy; and (14) clinical outcome. All of the patients included in this review were followed up as outpatients in the Nathan Smith Clinic and the Primary Care Clinic at Yale University School of Medicine and were typically followed up monthly at this stage of disease. Mortality data were obtained from inpatient or outpatient records.

Data are expressed as mean value ± SD of the mean. Comparisons were made using a Students t test. A p≤0.05 was considered significant.

RESULTS

Between 1991 and 1994, a total of 1,428 patients with HIV were admitted to the medical service. Of the 342 patients admitted with the diagnosis of pneumonia, pneumonitis, bronchitis, or PCP, 50 underwent diagnostic bronchoscopy. BAL fluid was positive for CMV in 17 of the 50 specimens. Of the 17 patients with a positive CMV culture, eight had BAL specimens positive for PCP and nine had no other pulmonary pathogens identified. The nine patients with CMV as the only isolate all fulfilled our criteria for a diagnosis of CMV pneumonitis, and it was these nine patients who composed the study group. No patient had evidence of Kaposi’s sarcoma.

Table 1—Characteristics of Patients Who Had Positive CMV Cultures and Characteristic Cytopathologic Conditions From Both BAL and TBB Specimens

<table>
<thead>
<tr>
<th>Patient No./Sex/Age, yr</th>
<th>Temperature, °C</th>
<th>Respiratory Rate</th>
<th>CD4</th>
<th>WBC × 10⁹</th>
<th>Po₂</th>
<th>LDH, IU/L</th>
<th>CMV Antigen</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/F/26</td>
<td>35</td>
<td>32</td>
<td>50</td>
<td>2.3</td>
<td>62</td>
<td>202</td>
<td>nd*</td>
</tr>
<tr>
<td>2/F/33</td>
<td>38.7</td>
<td>22</td>
<td>10</td>
<td>2.9</td>
<td>62</td>
<td>533</td>
<td>773</td>
</tr>
<tr>
<td>3/M/34</td>
<td>40.3</td>
<td>22</td>
<td>10</td>
<td>1.7</td>
<td>67</td>
<td>232</td>
<td>51</td>
</tr>
<tr>
<td>4/M/34</td>
<td>38.6</td>
<td>28</td>
<td>20</td>
<td>0.7</td>
<td>65</td>
<td>168</td>
<td>129</td>
</tr>
<tr>
<td>5/M/34</td>
<td>38.8</td>
<td>22</td>
<td>66</td>
<td>4.7</td>
<td>88</td>
<td>263</td>
<td>175</td>
</tr>
<tr>
<td>6/M/44</td>
<td>38.3</td>
<td>28</td>
<td>10</td>
<td>8.0</td>
<td>46</td>
<td>159</td>
<td>87</td>
</tr>
<tr>
<td>7/M/31</td>
<td>38.6</td>
<td>30</td>
<td>40</td>
<td>12.1</td>
<td>64</td>
<td>420</td>
<td>868</td>
</tr>
<tr>
<td>8/M/30</td>
<td>39.4</td>
<td>28</td>
<td>10</td>
<td>3.0</td>
<td>58</td>
<td>699</td>
<td>nd</td>
</tr>
<tr>
<td>9/M/37</td>
<td>39.4</td>
<td>26</td>
<td>50</td>
<td>2.6</td>
<td>45</td>
<td>1,050</td>
<td>400</td>
</tr>
</tbody>
</table>

*nd = no data.
Demographic and Clinical Characteristics

There was no significant difference in the demographic characteristics of the nine patients with CMV as the only identifiable pulmonary pathogen and the remaining patients with CMV coexisting with PCP (Table 1). There were seven men and two women, ranging in age from 26 to 44 years (mean, 33±5 years). The mode of acquiring HIV in the study population was not significantly different from the eight patients who also had PCP. Two patients (22%) were infected by same sex contacts, three (33%) acquired HIV through heterosexual contacts, and four (44%) were infected through IV drug use. There was no significant difference in the mean CD4 count in study patients and patients with multiple pathogens. The nine patients with positive CMV cultures all had CD4 counts less than 70 cells/mm³ and six had less than 30 cells/mm³ (mean, 29.6±22). All nine study patients had a history of prior opportunistic infections. Of the nine patients, none had either CMV retinitis or other active opportunistic infection at the time of presentation. Three of the nine study patients were treated with steroids at an equivalent dose of 60 mg of prednisone per day and empiric therapy for PCP (either trimethoprim/sulfamethoxazole or pentamidine). Both the steroids and empiric PCP therapy were discontinued after bronchoscopy did not demonstrate PCP.

The clinical characteristics and outcome of the study group are shown in Tables 1 and 2. Eight patients (89%) had fever and one was hypothermic. Nine patients (100%) were hypoxic on room air with a mean Po₂ of 62 mm Hg (±12). Although six patients (67%) had diffuse rales, three (33%) had normal results of pulmonary examination. All patients (100%) had an interstitial pattern on chest radiograph, and two (22%) had diffuse bilateral nodularity. Only one patient (11.1%) had an elevated WBC count and most were leukopenic (cell counts ranged from 0.7 to 3.8×10³/mm³ with a mean of 4.2×10³/mm³±3.6). LDH ranged from 159 to 1,050 IU/L (mean, 414±301). Two patients (22%) had normal LDH levels. In the seven patients in whom CMV antigen was measured, all had levels greater than 50 cells per 200,000 WBCs (Table 1).

Clinical Course and Outcome

Of the nine study patients, five were treated with ganciclovir (5 mg/kg every 12 h) for at least 14 days. All patients treated demonstrated improvement in cough, shortness of breath, and dyspnea on exertion. SaO₂ improved from 87 (±3) to 96 (±5), and chest radiographic abnormalities resolved in all patients. At 3 month follow-up, all five patients were asymptomatic with no pulmonary symptoms. At 6-month follow-up, three of the five patients continued to be asymptomatic. One had died of CNS lymphoma and the other had died of bacterial sepsis.

Of the nine patients identified in this study, four were not treated. These four patients were not treated because CMV pneumonia was not recognized as a treatable and distinct clinical entity at the time. Of these four patients, three died of respiratory failure. All three had autopsy confirmation of CMV pneumonia that showed dense infiltration of lung parenchyma and air spaces with inflammatory cells with CMV inclusion bodies. No other pathogens were identified. The one other nontreated patient continued to have persistent cough, dyspnea, and wheezing until she died one month after CMV diagnosis with disseminated Mycobacterium avium intracellulare.

Representative Case Reports

CASE 3

A 34-year-old HIV-positive man presented with recurrent nonproductive cough accompanied by

Table 2—Treatment and Outcome

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Treatment</th>
<th>Outcome</th>
</tr>
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<tbody>
<tr>
<td>1</td>
<td>Ganciclovir for 2 wk</td>
<td>Had resolution of symptoms, hypoxemia, and chest radiograph; did well without recurrence for more than 6 mo</td>
</tr>
<tr>
<td>2</td>
<td>Not treated</td>
<td>Continued to have symptoms of shortness of breath, cough, and dyspnea on exertion until she died 1 mo later</td>
</tr>
<tr>
<td>3</td>
<td>Ganciclovir for 2 wk</td>
<td>Had resolution of symptoms, hypoxemia, and chest radiograph; died 3 mo later of unrelated cause</td>
</tr>
<tr>
<td>4</td>
<td>Ganciclovir for 2 wk</td>
<td>Began on antiviral therapy after hospital discharge for CMV retinitis; had resolution of pulmonary symptoms and chest radiograph</td>
</tr>
<tr>
<td>5</td>
<td>Ganciclovir for 2 wk</td>
<td>Had a rapid resolution of symptoms, chest radiograph, and hypoxemia; was later begun on maintenance therapy for retinitis; did well for at least 6 mo</td>
</tr>
<tr>
<td>6</td>
<td>Ganciclovir for life</td>
<td>Had resolution of symptoms, hypoxemia, and chest radiograph; died 5 mo later of unrelated causes</td>
</tr>
<tr>
<td>7</td>
<td>Not treated</td>
<td>Died of respiratory failure</td>
</tr>
<tr>
<td>8</td>
<td>Not treated</td>
<td>Died of respiratory failure</td>
</tr>
<tr>
<td>9</td>
<td>Not treated</td>
<td>Died of respiratory failure</td>
</tr>
</tbody>
</table>
malaise, fevers, chills, rhinorrhea, and coryza. Medical history included CNS lymphoma and CD4 count less than 10 cells/mm$^3$. He had four previous hospital admissions for treatment of presumed but undocumented PCP within a 4-month period preceding this admission. He never had complete resolution of pulmonary symptoms. Medications included acyclovir, dexamethasone, erythropoietin, granulocyte colony-stimulating factor, didanosine, and aerosolized pentamidine. He had a temperature of 40.3°C with a heart rate of 124 beats/min and respiratory rate of 22 breaths/min. Physical examination was notable for mild thurlsh and clear lung fields. Laboratory data including a Po$_2$ of 67 mm Hg on room air, LDH of 233 IU/L, and WBC count of 1,700 cells/mm$^3$ with mild lymphocytosis and monocytosis. Chest radiograph revealed multiple parenchymal infiltrates and bilateral hilar fullness (Fig 1).

He was empirically started on a regimen of pentamidine and prednisone. Subsequent bronchoscopy was negative for acid-fast bacteria (AFB), PCP, and fungi. BAL and TBB specimen showed CMV inclusion bodies in the alveolar airspaces, epithelium, and interstitium with a mild inflammatory infiltrate. Cultures from BAL grew CMV. Electron microscopy of the transbronchial biopsy tissue was consistent with CMV pneumonitis. After these results were available, the pentamidine and prednisone therapy was stopped and ganciclovir therapy was started. His condition improved rapidly over the next week with resolution of hypoxemia and a marked decrease in interstitial markings on chest radiograph.

CASE 6

A 44-year-old HIV-positive man presented with a 1-week history of fevers to 38.8°C, fatigue, and lethargy. Medical history included PCP, adrenal insufficiency, and CD4 count less than 10 cells per cubic millimeter. Medications included hydrocortisone sodium succinate (Solu-Cortef) and trimethoprim/sulfamethoxazole. He had a temperature of 38.3°C, heart rate of 100 beats/min, respiratory rate of 25 breaths/min and O$_2$ saturation of 81% on room air. Physical examination revealed rales extending two-thirds up both lung fields, a systolic ejection murmur, and pitting edema. Laboratory data included a WBC count of 8,000 cells per cubic millimeter (57% segmented neutrophils, 35% bands), sodium of 122, serum bicarbonate of 17, LDH of 159 IU/L, and a room air Po$_2$ of 46 mm Hg. Chest radiograph showed bilateral interstitial infiltrates.

He was empirically started on a regimen of trimethoprim/sulfamethoxazole and high-dose steroids. His condition clinically deteriorated and on the third hospital day, bronchoscopy was performed. BAL revealed numerous viral inclusion bodies. TBB specimens demonstrated numerous viral inclusions with a mild inflammatory infiltrate. There was no evidence of PCP, AFB, or fungus. Cultures from BAL grew CMV. His CMV antigen level was 87 positive cells per 200,000 WBCs. Ganciclovir therapy was begun and his condition improved over the next 10 days with diminished symptoms, resolution of hypoxia, and marked improvement in pulmonary infiltrates.

CASE 8

A 30-year-old HIV-positive man presented with cough, shortness of breath, nausea, vomiting, and watery diarrhea. He had a CD4 count less than 10 cells per cubic millimeter. He had a temperature of 39.4°C, heart rate of 92 beats/min, and respiratory rate of 22 breaths/min. Physical examination revealed diffuse bilateral rales, and cool cyanotic extremities. Laboratory data included WBC count of 9,000 cells per cubic millimeter (85% segmented neutrophils, 9% bands), sodium of 132, and a Po$_2$ of 55 mm Hg on room air. Chest radiograph showed fluffy diffuse pulmonary infiltrates (Fig 2).

He was admitted to the ICU, intubated, and underwent bronchoscopy. BAL specimens were negative for AFB, bacterial, or fungal pathogens. Despite broad-spectrum antibiotics and steroids, he died within several days. Autopsy revealed diffuse patchy areas of mild consolidation in both lungs. Microscopic examination of his lungs revealed exten-

![Figure 1](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21742/)
sive evidence of CMV. Intranuclear and intracytoplasmic inclusions were identified in addition to an acute interstitial and focal organizing pneumonia with diffuse alveolar damage.

**DISCUSSION**

The spectrum of pulmonary disease in AIDS is a major source of morbidity and mortality, with more than 80% of patients having lung abnormalities during their disease course. Isolation of CMV from pulmonary secretions or lung tissue in patients with AIDS and pneumonia who undergo bronchoscopy and/or biopsy is common. However, there are few reported cases in which a clinical syndrome of dyspnea, hypoxemia, and diffuse interstitial infiltrates has been reported to correlate with histopathologic evidence of pulmonary CMV in the absence of other opportunistic infections. This study suggests that in at least some patients with AIDS, CMV represents a primary pulmonary pathogen.

Patients who met our inclusion criteria had CMV as the sole pulmonary pathogen as demonstrated by BAL and TBB specimens. All patients met the definition of AIDS with histories of multiple opportunistic infections and depletion of CD4 cell counts. There were no unique demographic characteristics in the study patients compared to patients with similar clinical presentations and multiple pulmonary pathogens.

We could not identify clinical characteristics that predicted CMV pneumonitis. Patients presented with nonspecific symptoms, including fever, shortness of breath, and dyspnea on exertion. Nearly all patients had an elevated LDH level while two patients had normal LDH levels. All patients had interstitial infiltrates on chest radiography. These nonspecific symptoms and signs are similar to other well-documented pulmonary infections in AIDS. Miles and coworkers found no association between CMV identified by BAL and hypoxemia, abnormal chest radiograph, or leukopenia. Other authors have documented nonspecific symptoms and signs in earlier studies as well.

We defined CMV pneumonitis in this study as the universal presence of the following three criteria; (1) positive CMV cultures from both BAL and TBB specimens; (2) characteristic cytopathic inclusion bodies from both BAL and TBB specimens; and (3) absence of any other pulmonary pathogen identified by bacterial, fungal, viral, or acid-fast stains or cultures. All of our patients (100%) had cytopathologic conditions consistent with CMV pneumonitis. One of the difficulties in reviewing prior investigations is that conclusions in many retrospective studies such as ours included patients who would not have met our criteria for CMV pneumonitis.

BAL has become the preferred procedure for diagnosing pulmonary infections in an immunocompromised host, largely because of the lack of predictive value of clinical characteristics alone. The diagnosis in patients with AIDS is often difficult because of the frequent occurrence of multiple pathogens. In bone marrow transplantation in which CMV pneumonitis is a frequent occurrence, the culture of virus from BAL is fairly reliable in identifying CMV infection. This has not been the case in HIV-infected patients, in whom recovery of CMV from BAL without cytopathologic evidence of invasion does not necessarily represent pneumonitis. The presence of viral antigens with cytopathologic changes in BAL in bone marrow transplant patients has shown a significant clinical correlation with active CMV disease and has been shown to be a specific marker for CMV infection. In all seven patients in whom it was measured, elevated CMV antigen levels were found. CMV antigen detection offers rapid diagnosis within hours, compared with days for cell culture, and is detectable for a longer period of time. This method needs to be studied prospectively but may offer a less invasive and more cost-effective means of diagnosing active disease.

Most of our patients were treated empirically for PCP with antibiotics and steroids. They generally had significant improvement in their symptoms with treatment. When steroid therapy was later tapered
or withdrawn, their symptoms returned. In fact, one patient (case 3) received five courses of anti-PCP therapy and steroids before going to bronchoscopy, which subsequently revealed only CMV and no evidence of PCP or other pathogens.

All of our patients who received anti-CMV therapy had dramatic improvement with resolution of clinical symptoms, improvement in objective signs, and, most profoundly, were able to be discharged from the hospital. All received a minimum of 2 weeks of therapy with ganciclovir. At 3 months of follow-up, all were asymptomatic without any further treatment. At 6 months of follow-up, three continued to do well with no further anti-CMV therapy. The remaining three patients died of other opportunistic infections. Other authors$^{17}$ have shown similar results in a small number of patients who were treated with anti-CMV therapy. However, only one of their patients would have met our criteria for CMV pneumonitis. It remains to be seen whether maintenance therapy with ganciclovir would be needed similar to therapy for CMV infections of other organs in this population.

In our study, four patients were not treated. Of these patients, three died within 2 weeks of presentation. The fourth patient continued to have pulmonary symptoms until she later died of unrelated causes. One study$^{15}$ showed an increased mortality when CMV is found on BAL in patients with AIDS. Although these patients did have multiple pathogens, the authors were unable to prove causal relationship. Millar and coworkers$^{7}$ suggested that CMV is not a lung pathogen in patients with AIDS. They identified six patients who had CMV alone, detected by BAL. Of their six patients, only three (50%) had cytopathologic changes. The patients in the study of Millar et al$^{7}$ were reported to have improved conditions without anti-CMV therapy. There are several reports in which CMV was documented as the sole pathogen in AIDS patients with pneumonitis.$^{18-23}$ Each of these reports involves only a small number of patients.

Because the diagnosis of CMV pneumonitis is one that is not established in our current medical literature, retrospective studies such as ours are extremely difficult to interpret since multiple authors define the clinical disease entity differently. We hope by presenting our definition of CMV pneumonitis with extremely explicit criteria, at least in this cohort, it will be clear exactly which patients had the potential of benefit from antiviral therapy.

In these nine patients, CMV appeared to have played a pathogenic role in clinical pneumonitis. In the few patients who were treated with specific antiviral treatment, clinical response was dramatic. The quality appeared to be improved as measured by the ability to leave the hospital, ability to travel to outpatient appointments, as suggested by information gleaned from outpatient progress notes. It was beyond the power of this study to determine if length of life was actually increased. To definitively answer these questions, a larger prospective study clearly needs to be done. In the absence of data from a randomized control trial, we recommend that clinicians consider this diagnosis in patients who present with clinical characteristics as strictly defined in our inclusion criteria and empirically treat with anti-CMV therapy.

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CHEST / 111 / 1 / JANUARY, 1997
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