result detected in the complete cohort. Recently, a systematic review identified a total of 13 studies that have described CMV infection in immunocompetent critically ill patients. CMV infection occurred in 0% to 36% of these critically ill patients. The authors found considerable heterogeneity in the methodology used to assess CMV infection and in the study population, which can explain the variability of the results. Our study has some limitations, including relatively low Acute Physiology and Chronic Health Evaluation (APACHE) II scores for each group, despite a relatively long length of stay in the ICU.

In conclusion, despite the use of a very sensitive PCR assay, we were unable to demonstrate a significant prevalence of CMV viremia in our immunocompromised ICU population. Accordingly, our data do not support empiric screening of nonimmunocompromised ICU patients in the absence of clinical evidence of CMV infection.

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Table 1—Patient Characteristics

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Group A</th>
<th>Group B</th>
<th>Summary</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>58.0 ± 17.6</td>
<td>46.5 ± 23.0</td>
<td>52.4 ± 21.0</td>
</tr>
<tr>
<td>Length of stay, d</td>
<td>19 ± 43</td>
<td>23 ± 11</td>
<td>20.9 ± 31</td>
</tr>
<tr>
<td>Admission unit</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trauma</td>
<td>17 (34)</td>
<td>22 (44)</td>
<td>39 (30)</td>
</tr>
<tr>
<td>General Surgery</td>
<td>14 (30)</td>
<td>10 (20)</td>
<td>24 (24)</td>
</tr>
<tr>
<td>Medical</td>
<td>9 (18)</td>
<td>10 (20)</td>
<td>19 (19)</td>
</tr>
<tr>
<td>Cardiovascular Surgery</td>
<td>9 (18)</td>
<td>8 (16)</td>
<td>17 (17)</td>
</tr>
<tr>
<td>APACHE II score</td>
<td>18 ± 9</td>
<td>20 ± 6</td>
<td>19 ± 8</td>
</tr>
<tr>
<td>Corticosteroids</td>
<td>15 (30)</td>
<td>18 (36)</td>
<td>33 (33)</td>
</tr>
<tr>
<td>Antibiotic</td>
<td>45 (90)</td>
<td>47 (94)</td>
<td>92 (92)</td>
</tr>
<tr>
<td>Duration, d</td>
<td>11.3 ± 8.2</td>
<td>18.0 ± 12.7</td>
<td>14.8 ± 11.0</td>
</tr>
<tr>
<td>Mechanical ventilation</td>
<td>44 (88)</td>
<td>45 (96)</td>
<td>92 (92)</td>
</tr>
<tr>
<td>Duration, d</td>
<td>10.4 ± 5.8</td>
<td>19.2 ± 12.3</td>
<td>15.1 ± 12.0</td>
</tr>
<tr>
<td>Mortality</td>
<td>7 (14)</td>
<td>7 (14)</td>
<td>14 (14)</td>
</tr>
</tbody>
</table>

Data are presented as mean ± SD or No. (%). Group A: baseline 5 d of ICU admission; Group B: baseline 10 d of ICU admission. APACHE = Acute Physiology and Chronic Health Evaluation.

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Author contributions: Dr Albert had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

Dr Gilbert: contributed to the study concept and design, acquisition of data, analysis and interpretation of data, drafting of the manuscript, and critical revision of the manuscript for important intellectual content.

Dr Rico: contributed to the study concept and design, analysis and interpretation of data, and critical revision of the manuscript for important intellectual content.

Dr Laglamme: contributed to the study concept and design, analysis and interpretation of data, and critical revision of the manuscript for important intellectual content.

Dr Albert: contributed to the study concept and design, acquisition of data, analysis and interpretation of data, drafting of the manuscript, and critical revision of the manuscript for important intellectual content.

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REFERENCES


Low-Dose Tissue Plasminogen Activator in Pulmonary Embolism

Benefit Remains Unclear

To the Editor:

We read the article in CHEST (February 2010) by Wang et al with great interest. However, we found several limitations. First, the authors tried to demonstrate the equivalency of low-dose and full-dose tissue plasminogen activator (tPA), although the superiority of full-dose tPA compared with anticoagulation in pulmonary embolism with right ventricular dysfunction is not firmly established, and its use remains controversial. The pulmonary embolism thrombolysis study, a large, ongoing European multicenter randomized controlled trial, will hopefully settle the controversy.

Second, the authors used a CT pulmonary angiography score as one of the surrogate end points. However, it is unclear whether...
this score is an adequate predictor of poor clinical outcome. To our knowledge, the correlation between this score and clinical outcome has never been validated in the setting of thrombolysis, and the use of surrogate end points in clinical research remains controversial. Third, the authors did not report the interobserver agreement (k value) in their study. Previous studies have found a low interobserver agreement for the assessment of the surrogate end points chosen. Lastly, the authors used a difference of 10 points in CT pulmonary angiography score to calculate the sample size, without providing the rationale for using such a difference. It is not clear whether such a difference is clinically relevant. Moreover, they did not specify the margins of noninferiority and nonsuperiority, and the study seems grossly underpowered to test the equivalence. The authors could have chosen the outcomes and calculated the sample size based on the best (but limited) available evidence. In an equivalence trial, both participants and outcome measures should be similar to those in the trial(s) that established the efficacy of the reference treatment (100 mg tPA, in this case).

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