pleura in situations such as the one described by Dr Kollef is not necessary. Pleuroscopy provides a much safer way, is much more informative, and enables direct inspection of the lesion with adequate biopsy whenever needed. Our experience with pleuroscopy for all kinds of pleural disease exceeds 500 procedures. The diagnostic potential is great, and the complication rate is extremely low. We had three instances of subcutaneous emphysema and a few cases of minor bleeding that did not necessitate transfusion.

In view of Dr Kollefs experience, I would advise him to keep our alternative in mind, and to insert a mediastinoscope, rather than a needle, through an intercostal space. It is easy and safe. For technical details I refer him, and other readers, to our earlier publications.1,4

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REFERENCES

To the Editor:

I appreciate the interest and comments of Drs Stark and Weissberg regarding the recent article describing loculated hemopneumothoraces of a major fissure in patients with COPD and associated pleural disease.4

Dr Stark’s chief concern is that the lesions described in this report actually represent infected subpleural bullae with air-fluid levels, and not loculated hemopneumothoraces within a major fissure. I feel that several points support the latter diagnosis. Both of the patients described had recent preceding chest radiographs that did not demonstrate evidence of bullae in the areas of concern. The new appearance of these cavity lesions over a short period of time (1 to 3 weeks) argues for the development of a new acute process, like a loculated hemopneumothorax. This is especially true in our second case with the large, oblong cavity following the location and position of the right major fissure.

The clinical presentation of our first case, with new onset of pleuritic chest pain and the finding of a new loculated hemopneumothorax adjacent to the infiltrate that had been present 1 week earlier, also favors a new process, like a loculated hemopneumothorax (which would also explain the patient’s symptoms).

In both cases, the loculated hemopneumothoraces were in direct contact with the major fissures superiorly and inferiorly, with the borders of the cavities tapered at the points where they communicated with the major fissure. In our first case, needle aspiration of the loculated hemopneumothorax allowed the migration of injected contrast out of the cavity into the major fissure, further supporting this diagnosis.

In the second case, needle aspiration of the loculated hemopneumothorax produced a tension pneumothorax. Upon reexpansion of the lung, the loculated hemopneumothorax with the air-fluid level had decompressed and emptied into the new pneumothorax, with the reexpanded lung showing no evidence of the prior loculated hemopneumothorax except for a new pleural effusion.

Finally, Peters et al describe infected bullae with air-fluid levels as usually occurring in the upper lobes of the lung.7 Our second case showed a lower lobe lesion, which—as in our first case—appeared to be within the major fissure on lateral chest radiograph and by computed tomography.

Thus, I agree with Peters et al that lung bullae with air-fluid levels are frequently noted in clinical practice but neglected in the medical literature.2 Loculated hemopneumothoraces are probably infrequent, and their further recognition in patients with combined parenchymal and pleural lung diseases may add to our understanding of these lesions.

Dr Weissberg recommends the use of pleuroscopy for the future evaluation and treatment of loculated hemopneumothoraces occurring within the fissures of patients with COPD and pleural disease. This recommendation is based on his experience with pleuroscopy4 and the serious complications of percutaneous needle drainage that occurred in two patients with this disorder.7 Loculated hemopneumothoraces occurring within a fissure appear to be rare.1 I agree with Dr Weissberg that pleuroscopy offers a potentially safer diagnostic and therapeutic alternative for these individuals. However, at the present time no documented experience exists regarding the use of pleuroscopy or methods other than needle drainage for this entity. Significant pleural adhesions or fibrosis would be expected to limit the efficacy of pleuroscopy, as would the need to place the mediastinoscope within a fissure to locate the fluid collections.

In the case of patients with known COPD and associated pleural disease who present with a cavitory lung lesion, loculated hemopneumothorax occurring within a fissure should be added to the differential diagnosis. Compatible radiographic findings may aid in differentiating these lesions from lung abscesses.1 If percutaneous needle aspiration of persistent loculations is performed, adequate facilities should be present to treat an emergent pneumothorax.1 Future experience with these lesions should clarify their natural history and the role of other drainage procedures like pleuroscopy.

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1 Kolleff MH. Loculated hemopneumothorax of a major fissure in patients with COPD and associated pleural disease. Chest 1990; 97:873-76
2 Stark P, Gadziala N, Greene R. Fluid accumulation in pre-existing pulmonary air spaces. AJR 1980; 134:701-06

Human Urinary Proteinase Inhibitor in the Treatment of P carinii Pneumonia

To the Editor:

Pneumocystis carinii pneumonia (PCP) remains a life-threatening opportunistic infection in patients with acquired immunodeficiency syndrome, collagen diseases, and malignancies treated with cytotoxic drugs and/or corticosteroids. We report the possible beneficial role of adjunctive human proteinase inhibitor in the treatment of PCP.

Patients' characteristics and treatment outcomes are shown in Table 1. Nine of the 15 patients had been treated with corticosteroids as a component of the chemotherapeutic regimens at the onset of PCP. Corticosteroids were immediately halted in these patients except for two patients with systemic lupus erythematosus (patients 7 and 9). All patients received conventional treatment for respiratory
Table 1—Patients’ Profiles and Treatment Outcomes

<table>
<thead>
<tr>
<th>Patient/Age (y)</th>
<th>Underlying Diseases</th>
<th>Chemo- or Immuno-therapy</th>
<th>PaO\textsubscript{2} on Dx (mm Hg)</th>
<th>Therapy for PCP</th>
<th>Interval to PaO\textsubscript{2} &gt;90 mm Hg (d)</th>
<th>Treatment Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/30</td>
<td>NHL</td>
<td>CHOP</td>
<td>84</td>
<td>TMP$MX$</td>
<td>—</td>
<td>Death (day 11)</td>
</tr>
<tr>
<td>2/47</td>
<td>ATL</td>
<td>CHOP</td>
<td>81</td>
<td>TMP$MX$ + m-PSL</td>
<td>—</td>
<td>Death (day 19)</td>
</tr>
<tr>
<td>3/34</td>
<td>ATL</td>
<td>CHOP</td>
<td>74</td>
<td>TMP$MX$ + PENT</td>
<td>24</td>
<td>Resolution</td>
</tr>
<tr>
<td>4/28</td>
<td>NHL</td>
<td>CHOP</td>
<td>64</td>
<td>TMP$MX$ + PENT</td>
<td>21</td>
<td>Resolution</td>
</tr>
<tr>
<td>5/54</td>
<td>ATL</td>
<td>VCR/PSL</td>
<td>59</td>
<td>TMP$MX$ + m-PSL</td>
<td>—</td>
<td>Death (day 12)</td>
</tr>
<tr>
<td>6/58</td>
<td>ATL</td>
<td>VP16/MTX/ADR</td>
<td>58</td>
<td>TMP$MX$ + PENT</td>
<td>—</td>
<td>Death (day 20)</td>
</tr>
<tr>
<td>7/34</td>
<td>SLE</td>
<td>PSL</td>
<td>55</td>
<td>TMP$MX$</td>
<td>10</td>
<td>Resolution</td>
</tr>
<tr>
<td>8/55</td>
<td>NHL</td>
<td>MACOP-B</td>
<td>68</td>
<td>TMP$MX$ + PENT</td>
<td>—</td>
<td>Death (day 10)</td>
</tr>
</tbody>
</table>

Median age, 54 Median age, 68

*Dx = diagnosis; NHL = non-Hodgkin lymphoma; ATL = adult T-cell leukemia; SLE = systemic lupus erythematosus; AML = acute lymphoblastic leukemia; AML = acute myelogenous leukemia; CHOP = cyclophosphamide, doxorubicin, vincristine, and prednisolone; VCR/PSL = vincristine and prednisolone; VP16/MTX/ADR = VP-16, methotrexate, and doxorubicin; MACOP-B = methotrexate, doxorubicin, cyclophosphamide, vincristine, prednisolone, and bleomycin; CA = cyclosporin A, BHAC-DMP = enocitabin, daunorubicin, 6-mercaptopurine, and prednisolone; TMP$MX$ = trimethoprim and sulfamethoxazole; m-PSL = high-dose methylprednisolone (20 mg/kg/d × 3 for days 1 and 2, 20 mg/kg on day 7 only for patient 13); PENT = pentamidine (4 mg/kg/d); PI = proteinase inhibitor, ulinastatin (20,000 U/d). †PaO\textsubscript{2} in patient 12 had already recovered to >90 mm Hg on day 16.

failure, including supplementary oxygen, intravenous fluid, nutritional support, and—where appropriate—ventilatory support. Specific antimicrobial therapy consisted of oral administration of trimethoprim-sulfamethoxazole (TMP-SMX), with additional intravenous pentamidine isothionate in patients 3, 4, and 5. Seven consecutive patients (patients 9 through 15) were treated with the adjunctive proteinase inhibitor, ulinastatin, in addition to oral treatment with TMP-SMX. They received 200 U/kg of ulinastatin (Miracle, Machida Pharmaceutical Co. Ltd., Tokyo, Japan) intravenously every 12 h until the oxygen pressure of arterial blood (PaO\textsubscript{2}) recovered to >90 mm Hg on room air. There was no significant difference in age or in the degree of hypoxia between the two groups of patients prior to PCP-directed therapies.

All seven patients treated with adjunctive ulinastatin recovered from PCP. They showed clinical improvement within 48 h after the start of therapy, which was demonstrated by an improvement in alveolar-arterial oxygen tension differences or PaO\textsubscript{2} levels and by a reduction of lung infiltrates on chest x-ray film. Ventilatory support was not required. The duration of ulinastatin treatment ranged from 7 to 16 days (median, 11 days). Pulmonary infiltrates on chest x-ray film completely disappeared in six of the seven patients, and no recurrence of PCP was seen throughout the course of the underlying diseases. No adverse effect, including the occurrence of liver damage, renal failure, bleeding diathesis, or exantheme, was observed. This contrasts with the observation that only three of the eight patients conventionally treated survived the disease. The cause of death in these five patients was attributed to a worsening of respiratory insufficiency due to PCP.

Ulinastatin is a potent and multihanded proteinase inhibitor purified from human urine. Its inhibitory effects have been demonstrated in granulocyte elastase, lysozymal thiol proteinase, and plasminogen activator. As a result, this agent can offer protection from tissue damage in acute pancreatitis and traumatic or burn shock. In PCP, proteinases released from tissue macrophages or circulating white blood cells are considered to be important mediators of pulmonary injury. Ulinastatin may suppress these overwhelming inflammatory processes, protect against tissue damage in the lung, and prevent irreversible acute respiratory failure. Although the number of patients was limited, these results suggest that adjunctive proteinase inhibitor is worthy of consideration for the treatment of PCP. A controlled clinical trial should be undertaken to clarify this issue.

Reprint requests: Dr. Akashi, First Department of Internal Medicine, Kyushu University, Fukuoka, Japan

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