Allergic bronchopulmonary aspergillosis (ABPA) is a disease of asthmatics that follows a protracted course. When ABPA is treated with high dose corticosteroids, it presents a difficult problem in clinical management. Five stages, based on clinical, roentgenographic, and immunologic criteria, have been identified as follows: (I) acute, (II) remission, (III) exacerbation, (IV) corticosteroid-dependent asthma, and (V) pulmonary fibrosis. We studied 24 ABPA patients actively followed for up to 11 years at our institution. We conclude that while there are no unique roentgenographic findings to define a particular stage, clinicoroentgenographic staging does aid in therapeutic management. Two major roentgenographic contributions are (1) to establish the diagnosis by demonstrating proximal bronchiectasis, and (2) to provide a baseline for an individual patient against which to monitor progressive changes and remissions.

S
ince 1952 when the first cases of allergic bronchopulmonary aspergillosis (ABPA) were diagnosed in England, there has been increased recognition of this disease entity.1 In 1968, the year that the first case of ABPA was reported in the United States, an English study found that up to 20 percent of asthmatic patients hospitalized with chronic pulmonary disability had ABPA.2 Since 1968, many adult and pediatric cases have been reported.3 Growing awareness of ABPA, definition of criteria for establishing the diagnosis, and understanding the natural history of this chronic pulmonary disease are responsible for the increased number of reported cases.

ABPA, treated with high dose corticosteroids, presents a difficult problem in clinical management. Diagnostic criteria associated with the disease are as follows: (1) asthma; (2) blood eosinophilia (>1,000/cu mm); (3) immediate skin reactivity to Aspergillus fumigatus (Af) antigen; (4) precipitating antibodies against the antigen; (5) elevated serum IgE concentration (>1,000 ng/ml); (6) history of pulmonary infiltrates; (7) central bronchiectasis; and (8) elevated serum levels of IgE and IgG antibodies to the antigen.3 Of these criteria, aside from No. 8, none is specific for ABPA with the exception of roentgenographically identified proximal bronchiectasis in the absence of cystic fibrosis which demonstrates proximal and distal bronchiectasis.

Transient and permanent manifestations of ABPA have been well described.4-7 Neither such transient changes as infiltrates and mucoid impactions, which reflect presence of active disease, nor all of the permanent changes are pathognomonic of this disease. Pulmonary fibrosis, blebs, and bullae occur in advanced ABPA but are nonspecific. The permanent changes diagnostic of ABPA are ectatic central bronchi

Table 1—ABPA Staging

<table>
<thead>
<tr>
<th>Stage</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>I. Acute</td>
<td>Patients are symptomatic: fever, bronchitis, sputum production, asthma. Laboratory: elevation of serum IgE, blood eosinophilia Roentgenographic: pulmonary infiltrates may be present Management: corticosteroids to achieve remission</td>
</tr>
<tr>
<td>II. Remission</td>
<td>Patients are asymptomatic. Laboratory: decline in IgE and blood eosinophilia Roentgenographic: resolution of acute pulmonary infiltrates Management: maintenance corticosteroids not required</td>
</tr>
<tr>
<td>III. Exacerbation</td>
<td>Patients may show acute symptoms or may be asymptomatic Laboratory: doubling of remission IgE level in asymptomatic patients and increased IgE levels in symptomatic patients Roentgenographic: appearance of new findings, ie, infiltrates, mucoid impaction, etc. Management: induction of remission with corticosteroids: then maintenance corticosteroids are not required.</td>
</tr>
<tr>
<td>IV. Corticosteroid dependent asthma</td>
<td>Symptoms: Patients are symptomatic from asthma requiring long-term corticosteroid therapy Laboratory: elevation of IgE levels Roentgenographic: variable Management: long-term corticosteroids</td>
</tr>
<tr>
<td>V. Fibrosis</td>
<td>Symptoms: dyspnea and other manifestations of fibrotic lung disease Laboratory: irreversible obstructive and or restrictive PFTS IgE levels variable (elevation of IgE may reflect continued activity) Roentgenographic: pulmonary fibrosis Management: long-term corticosteroids</td>
</tr>
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with peribronchial thickening seen on end as ring shadows or in tangent as parallel-line shadows.

As more cases of ABPA have been recognized, certain clinicoroentgenologic patterns have emerged. The observed patterns have recently been organized into a clinical staging system. This staging system aids in management of this patient group that is ordinarily difficult to treat. The radiologist must be aware of this disease entity and its roentgenographic manifestations, both minimal and severe. Further, knowledge of response to therapy and the clinical staging of this disease enables the radiologist to play a more active role in the care of these patients. On the basis of clinical, laboratory, and roentgenographic findings, five stages of the disease have been established: (I) acute, (II) remission, (III) exacerbation, (IV) corticosteroid-dependent asthma, and (V) pulmonary fibrosis (Table 1).

Because of the difficulty in establishing the diagnosis of ABPA before significant bronchiectasis has occurred, patients may present in more advanced stages. In addition, despite appropriate diagnosis, staging, and therapy, some patients presenting in stage

![Image](http://journal.publications.chestnet.org/pdaccess.ashx?url=/data/journals/chest/21459/)

**Figure 1.** A 16-year-old white boy with a long history of asthma presented with an exacerbation of his disease. X-ray film of 7/19/77 demonstrates lingular collapse presumably secondary to mucoid plugging of the lingular bronchi.

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Age</th>
<th>Presentation Date</th>
<th>Presentation Stage</th>
<th>Initial IgE (ng/ml)</th>
<th>Roentgenographic Findings</th>
<th>Current Stage</th>
<th>Current IgE (ng/ml)</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>12</td>
<td>1976</td>
<td>I</td>
<td>5770</td>
<td>+</td>
<td>IV</td>
<td>7710</td>
<td>Migratory infiltrates</td>
</tr>
<tr>
<td>2</td>
<td>41</td>
<td>1982</td>
<td>I</td>
<td>1441</td>
<td>+</td>
<td>IV</td>
<td>627</td>
<td>Chest x-ray stable</td>
</tr>
<tr>
<td>3</td>
<td>29</td>
<td>1983</td>
<td>I</td>
<td>12457</td>
<td>CXR + Tomo</td>
<td>II</td>
<td>5018</td>
<td>Tomography required to establish presence of proximal bronchiectasis</td>
</tr>
<tr>
<td>4</td>
<td>32</td>
<td>1983</td>
<td>I</td>
<td>8880</td>
<td>+</td>
<td>IV</td>
<td>1905</td>
<td>CXR: Massive homogeneous consolidation— responded well to corticosteroid Rx.</td>
</tr>
<tr>
<td>5</td>
<td>31</td>
<td>1975</td>
<td>I</td>
<td>13920</td>
<td>+</td>
<td>II</td>
<td>840</td>
<td>Bilateral upper lobe infiltrates</td>
</tr>
<tr>
<td>6</td>
<td>14</td>
<td>1975</td>
<td>I</td>
<td>7440</td>
<td>+</td>
<td>II</td>
<td>2350</td>
<td>Bilateral upper lobe infiltrates</td>
</tr>
<tr>
<td>7</td>
<td>24</td>
<td>1977</td>
<td>I</td>
<td>3600</td>
<td>+</td>
<td>IV</td>
<td>206</td>
<td>Bilateral upper lobe infiltrates</td>
</tr>
<tr>
<td>8</td>
<td>24</td>
<td>1977</td>
<td>I</td>
<td>15000</td>
<td>+</td>
<td>IV</td>
<td>3680</td>
<td>Right middle lobe infiltrate</td>
</tr>
<tr>
<td>9</td>
<td>11</td>
<td>1982</td>
<td>I</td>
<td>5772</td>
<td>+</td>
<td>III</td>
<td>11084</td>
<td>Right middle lobe infiltrate (CT scan)</td>
</tr>
<tr>
<td>10</td>
<td>52</td>
<td>1978</td>
<td>I</td>
<td>3500</td>
<td>+</td>
<td>III→IV</td>
<td>1233</td>
<td>Right middle lobe infiltrate (CT scan)</td>
</tr>
<tr>
<td>11</td>
<td>16</td>
<td>1977</td>
<td>I</td>
<td>4150</td>
<td>+</td>
<td>III→IV</td>
<td>1184</td>
<td>—Migratory infiltrates —During flares, IgE rose to 6250 —Bronchogram with bronchiectasis on opposite side</td>
</tr>
<tr>
<td>12</td>
<td>40</td>
<td>1978</td>
<td>I</td>
<td>3614</td>
<td>+</td>
<td>V</td>
<td>1554</td>
<td>Extensive upper lobe fibrotic changes</td>
</tr>
<tr>
<td>13</td>
<td>23</td>
<td>1979</td>
<td>I</td>
<td>18831</td>
<td>+</td>
<td>III→IV</td>
<td>165</td>
<td>—LUL infiltrate —During flare (1/26/81) IgE rose to 2621</td>
</tr>
<tr>
<td>14</td>
<td>27</td>
<td>1972</td>
<td>I</td>
<td>8880</td>
<td>+</td>
<td>III</td>
<td>3033</td>
<td>RML infiltrate 5/10/83</td>
</tr>
<tr>
<td>15</td>
<td>14</td>
<td>1978</td>
<td>I</td>
<td>9954</td>
<td>+</td>
<td>III→IV</td>
<td>12026</td>
<td>RUL infiltrate</td>
</tr>
<tr>
<td>16</td>
<td>17</td>
<td>1979</td>
<td>I</td>
<td>394</td>
<td>+</td>
<td>V</td>
<td>169</td>
<td>Atypical mycobacterial infection with dissemination following bronchogram at outside institution</td>
</tr>
</tbody>
</table>

Table 2—Patient Summaries
I will progress to stages III, IV, and V (Fig 1). Appropriate corticosteroid therapy can often achieve episodic remission. Long-term effectiveness of this therapeutic modality in either retarding or halting the progressive destruction of pulmonary parenchyma has yet to be established clearly. 

**MATERIALS AND METHODS**

Between February 1967 and the present, 62 patients with proven ABPA were evaluated at our institution. These patients were nearly evenly divided between males and females. Their ages varied from five years of age to 66 years. All of these patients were evaluated with appropriate roentgenographic and immunologic tests.

The chest roentgenogram and bronchography or tomography were utilized to demonstrate ABPA findings. In selected patients, a questionably abnormal roentgenogram led to bronchography in the past. Bronchographic evaluation was replaced by tomography after several patients experienced adverse effects to the bronchographic procedure.

While a large number of immunologic tests are used to detect sensitivity to *Aspergillus fumigatus* and ABPA, serum IgE levels and precipitating antibodies to *A. fumigatus* are most useful. These tests are performed on all patients at presentation. Following diagnosis, monthly IgE levels are obtained.

Additionally, the baseline level of blood eosinophilia may be determined. Pulmonary function testing (PFT) is performed during the course of their disease, but since PFTs were found to contribute little to clinical management, they are not required in the management protocol.

The protocol for following patients with ABPA advocated by the Allergy Service at our institution calls for chest roentgenograms every three months during the first year following diagnosis, and yearly thereafter. In addition, with any doubling of IgE levels, a chest x-ray film is obtained to exclude an infiltrate occurring during any exacerbation.

**FIGURE 2.** Patient as in Figure 1. On 7/22/77, there is demonstrated further mucoid impaction with involvement of all segments of the left lung. Because of an elevated serum IgE, corticosteroid therapy was initiated.

**FIGURE 3.** Patient as in Figure 1. Five weeks later, on 9/8/77, the patient was in remission with all acute changes having resolved. No plain film findings at this time are seen in the left side of the chest. A few cystic changes are seen in right infralobar region (arrows).

To determine the role of roentgenographic studies in initial staging and long-term management of patients with ABPA, we reviewed the diagnostic examinations and clinical records of 24 of the 62 patients who have been actively followed at our institution from 1972 to the present. Patients followed at other institutions, lost to follow-up, or deceased have been excluded.

**FIGURE 4.** Patient as in Figure 1. Bronchography establishes the diagnosis of proximal bronchiectasis involving middle lobe bronchi. The mucoid impaction was a result of the patient's asthma, not reflecting the presence of proximal bronchiectasis.
RESULTS

Of the 24 patients in this series (Table 2), the largest group, 16, presented with stage I, acute disease. Two patients were classified as stage II, remission phase, upon presentation, and six patients were in stage IV, corticosteroid-dependent ABPA. The stage I patients achieved remission, with three of the 16 sustaining remission and remaining in stage II. The other 13 patients presenting in stage I progressed to more advanced stages with two classified as stage III, four in stages III to IV, and five in stage IV. Two patients presenting in the first stage developed pulmonary fibrosis (stage V).

Six patients had corticosteroid-dependent asthma (stage IV) when their management began. Two of these patients progressed to stage V, while in four, ABPA was controlled with high dose corticosteroid therapy.

The length of stay in a particular stage varied with the individual patient, as did progression of ABPA. Neither the time course nor advancement of the disease followed a predictable pattern in those patients followed for up to 11 years. The necessity for high dose corticosteroid therapy to control ABPA flares was not a prognostic indicator for development of pulmonary fibrosis. Predisposing factors for development of pulmonary fibrosis within this patient population are not as yet known.

DISCUSSION

From our review of the sequential examinations of patients with ABPA, we have observed the following roentgenographic changes: proximal bronchiectasis, perhaps the most important finding, tramline and linear shadows, and ring shadows are permanent manifestations. Mucoid impactions and infiltrates appear transiently and do resolve (Fig 1 through 3). In stages I through IV, and possibly V, the presence of mucoid impactions and infiltrates is evidence for active disease requiring treatment in patients whose chest roentgenograms and/or tomograms exhibit proximal bronchiectasis, the roentgenographic sign of ABPA (Fig 3 through 7).

The following trends were observed: stage I patients with plain film findings of ABPA responded to therapy by clearing of the migratory infiltrates and areas of

FIGURE 5. An 8-year-old boy with long-standing asthma. A questionable area of bronchiectasis was noted in the superior margin of the right hilus (arrow).

FIGURE 6. Patient as in Figure 5. Before high dose corticosteroid therapy was instituted, tomograms through both perihilar regions were obtained. Striking bronchiectasis of the right upper lobe and the origin of the right middle lobe (arrows) is seen.

FIGURE 7. Patient as in Figure 5. While this patient progressed clinically to stage IV (corticosteroid-dependent asthma), his roentgenographic findings remain stable over four years.
mucoid impaction (Fig 1 through 3). While predomi-
nance of upper lobe involvement is clear, the lower
lobes are not infrequently affected. The residua of
mucoid impactions frequently remain in the lungs
manifested by parallel and tramline shadows, as well as
ring shadows. These are indicative of bronchiectasis
(Fig 6).

In stage II, since none of the active manifestations is
present (ie, mucoid impaction, tooth paste shadows,
etc), the residual changes may be extremely subtle,
requiring either tomography or bronchography to
establish their presence. Classification of patients in
stage II by PA roentgenograms obviates the necessity
for further roentgenographic evaluation since pres-
ence of ABPA has already been established.

In stage III, (recurrent exacerbation), new infiltrates
frequently develop. In a few patients, in addition to
areas of new infiltrate, new areas of mucoid impaction
and impaction of previously damaged bronchi may be
seen. As in stage I, the infiltrates predominate in the
upper lung fields and are frequently more central than
pneumonias unassociated with cosinophilia. The ma-
nority of patients in stage III will soon progress to stage
IV, although some patients may return to the second
stage. No certain prognostic indicators for progression
or regression of the disease process have been identi-
fied.

In stage IV, corticosteroid-dependent ABPA, the
findings are indistinguishable roentgenographically
from those of stage II patients. Clinically, these pa-
tients require corticosteroid treatment for control of
asthma. The dose of corticosteroid necessary for con-
tral of asthma may be less than that needed to prevent
new roentgenographic infiltrates from ABPA.

Stage V is clinically present when there is extensive
fibrotic change and irreversible obstructive lung dis-
ase based on pulmonary function testing. Neither
pulmonary lesions (fibrotic changes) of PFTs are re-
versed with high dose steroid therapy, although corticosteroids are required for ABPA flares.9 No consistent roentgenographic trends are observed in
every case.

Because of variability in duration of ABPA prior to
treatment, there is no precise roentgenographic cor-
relation with each of the clinical stages. In those stage I
patients in whom the diagnosis of ABPA is established
early and the treatment effective, only the most
minimal changes of ABPA may be identifiable. In
patients diagnosed only after unrecognized but re-
peated bouts of untreated ABPA more extensive pul-
monary changes are found.

The presence of infiltrates on chest roentgenograms
creates with acute and recurrent exacerbations of
ABPA (stages I and III). The presence of fibrotic
changes on roentgenograms which are present in stage
V indicates that irreversible pulmonary damage has
occurred but does not correlate well with extent of PFT
abnormalities.

While roentgenographic severity does not reflect
the clinical stage of ABPA predictably, roentgeno-
graphic findings are quite useful in establishing a
baseline for each individual patient and in following
the patient's response to treatment. Further, when
exacerbations occur, as evidenced by clinical symp-
toms and evaluation of serum IgE, confirmatory roen-
tgenographic findings, such as mucoid impactions and
infiltrates, reflect the active disease process.

Conclusions
ABPA is a disease with protracted course. The
affected patient population is comprised of asthmatics,
and ABPA flares may resemble attacks of uncompli-
cated asthma. ABPA, however, may cause permanent
pulmonary changes (ie, bronchiectasis and fibrosis).
While the clinical management of ABPA is difficult and
progressive disease is common, we believe that the
opportunity for early diagnosis and treatment is impor-
tant to prevent irreversible pulmonary destruction.10
Clinicoroentgeographic staging is therefore helpful in
the therapeutic management of these patients.

Roentgenographic findings of ABPA seen acutely in
stages I, III, and IV include not only proximal bron-
chiectasis, the sine qua non of the diagnosis in absence
of cystic fibrosis, but also mucoid impaction and
pulmonary infiltrates, often in an upper lobe and
central distribution. Stage V patients exhibit pulmo-
ary fibrosis upon which the acute changes may also be
superimposed.

While there is no diagnostic roentgenographic find-
ing to define a particular stage, there are two major
roentgenographic contributions to managing these
ABPA patients: first, to establish the diagnosis initially
by demonstrating proximal bronchiectasis on plain
film, tomography, or bronchography; and, second, to
provide a baseline for each individual patient against
which to monitor remissions and progressive changes.

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The registration fee is $165.00 and will include the Art Institute Reception and Awards Luncheon. An additional fee of $5.00 will be charged for the Nurses' Luncheon. To receive further information please contact: Conference Headquarters, 2121 Wisconsin Avenue, NW, Washington, DC 20007 (202) 944-3176.