Table 1—Aspergillus terreus Infections with Pulmonary Involvement

<table>
<thead>
<tr>
<th>Authors</th>
<th>Age/Sex</th>
<th>Underlying Disease</th>
<th>Risk Factors</th>
<th>Type of Lung Involvement</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kennedy et al.</td>
<td>54/M</td>
<td>Ankylosing spondylitis</td>
<td>Fibrosis and cavitation</td>
<td>Pneumonitis</td>
</tr>
<tr>
<td>Laham et al</td>
<td>58/M</td>
<td>Tuberculosis</td>
<td>Corticosteroids</td>
<td>Mycetoma</td>
</tr>
<tr>
<td></td>
<td>18/M</td>
<td>None</td>
<td>None</td>
<td>Allergic bronchopulmonary aspergillosis</td>
</tr>
<tr>
<td>Vincken et al</td>
<td>43/F</td>
<td>None</td>
<td>None</td>
<td>Allergic bronchopulmonary aspergillosis</td>
</tr>
<tr>
<td></td>
<td>57/F</td>
<td>None</td>
<td>None</td>
<td>Allergic bronchopulmonary aspergillosis</td>
</tr>
<tr>
<td>Tracy et al</td>
<td>23/M</td>
<td>Acute non-lymphocytic leukemia</td>
<td>Chemotherapy-induced neutropenia and broad-spectrum antibiotics</td>
<td>Invasive pulmonary aspergillosis</td>
</tr>
<tr>
<td>Chang and King</td>
<td>60/M</td>
<td>Acute myelomonocytic leukemia</td>
<td>Chemotherapy-induced neutropenia and broad-spectrum antibiotics</td>
<td>Invasive pulmonary aspergillosis</td>
</tr>
<tr>
<td>Moore et al (present case)</td>
<td>70/F</td>
<td>Nodular poorly differentiated lymphocytic lymphoma</td>
<td>Chemotherapy and corticosteroids</td>
<td>Invasive pulmonary aspergillosis</td>
</tr>
</tbody>
</table>

of involving the lungs. Its manifestations span the spectrum of pulmonary disease caused by *A. fumigatus* (Table 1). Three cases of allergic bronchopulmonary aspergillosis caused by *A. terreus* have been reported. There is a single report of a mycetoma occurring in the setting of prior tuberculosis, and another case of apparent saprophytic lung involvement in a patient with ankylosing spondylitis. The first case of invasive pulmonary aspergillosis secondary to *A. terreus* was reported by Tracy et al in 1983. Multiple pulmonary nodules developed in the setting of acute non-lymphocytic leukemia, chemotherapy-induced neutropenia and broad-spectrum antibiotics. Open lung biopsy revealed invasive *A. terreus* infection. Subsequent autopsy confirmed pulmonary nodules (some cavitating), vascular invasion with secondary infarction, and involvement of several other organs (including brain, thyroid and kidney). The second case, reported by Chang and King in 1986, involved a 60-year-old patient with acute myelomonocytic leukemia, neutropenia and recent broad-spectrum antibiotic therapy. This patient developed a left upper lobe opacity which cavitated with development of an "air-crescent sign." Bronchoscopy confirmed invasive infection with *A. terreus*.

Invasive pulmonary aspergillosis caused by *A. fumigatus* and *A. flavus* occurs predominantly in the setting of hematologic malignancy, neutropenia and broad-spectrum antibiotic therapy. That this same group of patients is at risk for invasive *A. terreus* pulmonary infection has been documented in the two patients previously reported. The case reported here is the first in a patient with lymphoma and in the absence of neutropenia and broad-spectrum antibiotic therapy. However, our patient was on corticosteroid therapy, another recognized risk factor for the development of invasive disease caused by other Aspergillus species.

Studies in various areas of the United States have yielded divergent findings regarding the prevalence of *A. terreus* in the air and in clinical specimens. In any case, *A. terreus* can no longer be immediately discounted as a laboratory contaminant in pulmonary specimens, especially in immunosuppressed patient populations. The spectrum of pulmonary disease associated with *A. fumigatus* and *A. flavus* can also be caused by *A. terreus*. Despite therapy with amphotericin B, all three reported patients with invasive *A. terreus* pulmonary infection have died with evidence of persistent fungal disease.

References

10. Solomon WR, Burge HF, Boise JR. Airborne *Aspergillus fumi-
gatus* levels outside and within a large clinical center: J Allergy Clin Immunol 1978; 62:56-60

Heyde's Syndrome*

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We describe a patient with Heyde's syndrome in whom upper gastrointestinal angiodysplasias were demonstrated and who was successfully treated by bioprosthetic aortic valve replacement. (Chest 1988; 94:891-92)

There has been much recent interest in the uncommon association of calcific aortic valvular disease with chronic gastrointestinal bleeding (Heyde's syndrome), and the role of aortic valve replacement as the definitive treatment of this condition.1-4 We have recently encountered one patient with this syndrome and believe that two particular aspects of this case deserve mention: its association with angiodysplasias of the upper gastrointestinal tract and the efficacy of bioprosthetic aortic valve replacement.

CASE REPORT

A 68-year-old woman presented initially with acute congestive heart failure and was found on examination to have aortic stenosis and incompetence. Coronary angiography confirmed the diagnosis, and a gradient of 60 mm Hg across the calcified aortic valve was recorded. She had a six-year history of unexplained anemia (normocytic, normochronic) that had been extensively investigated on several occasions with hematologic studies, including bone marrow examination, fecal occult blood screening, and gastrointestinal endoscopy, and was being treated by her family doctor with periodic blood transfusions (one or two units of packed red blood cells every three months on average). She remained otherwise well, and her hemoglobin on admission was 100 dl⁻¹ with a platelet count of 543 x 10⁹ L⁻¹. All other blood test results, including prothrombin time, partial thromboplastin time, iron studies, etc., were within normal limits.

Two days following cardiac catheterization, she produced coffee-ground vomit, and the hemoglobin fell 25 gl⁻¹. Gastroscopy demonstrated two significant, nonbleeding, angiodysplastic lesions in the prepyloric region that were sclerosed successfully. The patient refused additional invasive investigation, exhibited no further bleeding, and was referred to us for treatment of her symptomatic valvular lesion. The calcified, stenosed aortic valve was replaced with a 19-mm Ionescu-Shiley prosthesis, and the patient was discharged with a hemoglobin of 110 dl⁻¹ one week postoperatively. A hemoglobin of 112 dl⁻¹ was recorded two months postoperatively, and the patient had not required blood transfusion in the interim.

DISCUSSION

This case report is a typical illustration of Heyde's syndrome, that of calcific aortic valve disease and anemia presumed to be secondary to occult gastrointestinal bleeding from vascular ectasias, and also shows that such lesions may occur in the upper gastrointestinal tract. This uncommon site should therefore be considered if angiodysplasias of the colon, the usual site of such lesions,4 are not demonstrated. Indeed, a recent review of over 90 cases demonstrated angiodysplasias in the upper gastrointestinal tract (duodenum) of only one patient,5 most lesions occurring distally. The fact that gastric angiodysplasias were detected in this case only after a significant upper gastrointestinal bleed, and not over a period of six years of occult bleeding, is in keeping with the spectrum of presentation of such lesions.4

Although the mechanism of bleeding and its association with the aortic valvar disease is still debated, resolution of gastrointestinal bleeding following aortic valve replacement led to the recommendation that valve replacement be the definitive treatment for both components of the syndrome.1-4 A bioprosthesis would seem to be advantageous, as postoperative anticoagulation would then be avoided.4 This has been confirmed by a recent report in which postoperative gastrointestinal bleeding stopped after a mechanical valve was replaced with a bioprosthesis.5 Although our follow-up is not yet complete, early indications suggest that aortic valve replacement may be of value in the management of this patient.

ACKNOWLEDGMENT: We wish to thank Heather Motloch for her help in the preparation of this paper.

REFERENCES