A CASE OF GASTROINTESTINAL BLEEDING ASSOCIATED WITH CALCIFIC AORTIC STENOSIS

Takahide Ito, M.D. and Hironobu Ando, M.D.

Gastrointestinal bleeding is not rare in patients with calcific aortic stenosis (AS) in Western countries, which has suggested an association between the two conditions. However, there has been no previous report of this association in Japan.

In this report, we describe a patient with AS who had recurrent gastrointestinal bleeding. An 84-year-old female who had been followed for severe AS presented with recurrent rectal bleeding, but the site of bleeding could not be detected despite various examinations, including endoscopy. She died of progressive heart failure during hospitalization. Postmortem examination disclosed severe left ventricular hypertrophy with calcific aortic stenosis. Microscopically, angiodysplastic lesions were observed in the submucosa throughout the gastrointestinal tract.

In 1958, Heyde\textsuperscript{1} and later Schwarz\textsuperscript{2} suggested an association between aortic stenosis (AS) and gastrointestinal bleeding. Since then, numerous studies have confirmed this association and have suggested that angiodysplasia may account for the bleeding. In Japan, however, there have been no previous reports regarding this association. In this report, we describe a patient with AS who suffered from repeated gastrointestinal bleeding due to angiodysplasia that was confirmed by postmortem examination.

CASE REPORT

An 84-year-old female, who had been followed up for angina pectoris due to AS from the age of 79 years, and who had refused valve replacement, presented with a 2-month history of recurrent, painless rectal bleeding. A barium enema at that time revealed only diverticulae of the ascending colon. Thereafter, she continued to experience recurrent rectal bleeding for which she did not receive any treatment. On the day of admission, she complained of increasing chest pain and dyspnea on effort. Routine blood tests revealed anemia (Hb:8.1 g/dl). On physical examination, she appeared pale and unwell. The pulse rate was 84/min and regular, the respiration rate was 18/min, and the blood pressure was 120/86 mmHg. On auscultation, S1 and S2 were normal; S4 was heard at the apex; a grade 4/6 ejection mur-

<table>
<thead>
<tr>
<th>TABLE I HEMODYNAMIC DATA AT CARDIAC CATHETERIZATION</th>
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<tbody>
<tr>
<td><strong>Pressure (mmHg)</strong></td>
</tr>
<tr>
<td>Right atrium</td>
</tr>
<tr>
<td>Right ventricle</td>
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<tr>
<td>Pulmonary artery</td>
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<tr>
<td>Pulmonary capillary wedge</td>
</tr>
<tr>
<td>Left ventricle</td>
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<tr>
<td>Aortic</td>
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<tr>
<td>Cardiac (l/l/min) output</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>2</td>
</tr>
<tr>
<td>22/2 end-diastolic</td>
</tr>
<tr>
<td>26/10 mean</td>
</tr>
<tr>
<td>6</td>
</tr>
<tr>
<td>320/0 end-diastolic</td>
</tr>
<tr>
<td>145/55 mean</td>
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<td>3.05</td>
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</tbody>
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Key words:
- Calcific aortic stenosis
- Gastrointestinal bleeding
- Angiodysplasia

(Received November 4, 1993; accepted January 27, 1994)

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Fig. 1. Macroscopic findings of the heart
Left: Excised myocardium at the papillary muscle level, showing marked concentric hypertrophy of the left ventricle (wall thickness: 2.0 cm)
Right: Appearance of the aortic valve (arrow). Note the rigid valve with a narrow orifice.

...mur radiating to the neck was heard at the right sternal border and there were fine crepitations at the bases of both lungs. The liver was palpable 2 finger-breaths below the right costal margin. No peripheral edema was noted and neurological examination revealed no abnormalities. An electrocardiogram showed normal sinus rhythm and left ventricular hypertrophy with poor R wave progression in the right precordial leads. Chest X-ray showed marked cardiomegaly (CTR: 70%) and mild pulmonary congestion. A cardiac ultrasonographic examination showed severe left ventricular hypertrophy with normal systolic function and thickening of the aortic valve. A Doppler examination revealed an estimated aortic pressure gradient of 194 mmHg. Hematological examination revealed normochromic, normocytic anemia (Hb: 8.1 g/dl, Ht: 25%). Biochemistry tests showed a slightly elevated blood urea nitrogen level (29 mg/dl, normal: 8–18), but were otherwise normal. Hemostatic examinations, including the bleeding time and the prothrombin time, were all within normal limits. Cardiac catheterization performed on the 18th hospital day revealed a mean pressure gradient of 151 mmHg across the aortic valve and an estimated aortic valve area of 0.26 cm². Other hemodynamic data are shown in Table I. A coronary angiographic examination showed normal coronary arteries, and a left ventricular angiographic examination disclosed markedly and diffusely thickened ventricular wall and normal systolic function. Fluoroscopically, severe calcification was observed around the aortic valve.

After admission, oral intake was stopped and 3 packs of red blood cells were transfused, resulting in the transient cessation of rectal bleeding. However, slight bleeding recurred a few days after the initiation of oral intake. Endoscopic examination of the upper and lower gastrointestinal tract did not detect any origin of the bleeding. Subsequently, selective mesenteric arteriography also failed to locate the origin of the bleeding. During the second colonoscopic examination on the 76th hospital day, the sigmoid colon was accidentally perforated and an emergency laparotomy was performed. The operation was completed successfully, but on the 10th postoperative day she developed overt heart failure which was resistant to any treatment. Her condition worsened and she died several days later.

The postmortem examination showed marked hypertrophy of the left ventricle (Fig. 1, Left) with aortic valve thickening and a lack of mobility due to calcium deposits (Fig. 1, Right). Microscopic examina-
tion of a specimen from the ascending colon (Fig. 2, Left) revealed multiple clusters of irregular vessels in the submucosa. Similar lesions were observed in other areas, including the small intestine, the stomach and the esophagus (Fig. 2, Right).

DISCUSSION

We occasionally encounter elderly patients with recurrent unexplained gastrointestinal bleeding. Approximately one-fourth of such patients in Western countries reportedly have concurrent aortic stenosis (AS), which has suggested that gastrointestinal bleeding may be associated with AS. Although there is still some controversy on this point. The lesion that is thought to cause the bleeding, angiodyplasia, was first recognized at autopsy by Boss and Rosenbaum in 1971. Angiodyplasia is mainly present in the ascending colon but it can also occur in other parts of the gastrointestinal tract. This condition is difficult to detect by usual gastrointestinal procedures, such as endoscopy and barium studies, since it is usually covered by a thin layer of mucosa. Selective mesenteric arteriography has been shown to be the most useful method for detecting this condition.

Although the etiology of angiodyplasia remains unclear, these lesions are detected in over 50% of elderly individuals, suggesting that they may develop as part of the aging process. In patients with AS, however, angiodyplasia bleeds more often than in controls implying that the stenotic valve may facilitate bleeding from angiodyplastic lesions. Boley et al. postulated that the low perfusion pressure associated with AS may cause bleeding by promoting ischemic necrosis of preexisting angiodyplastic vessels. Other investigators have proposed that calcific AS may produce coagulation defects severe enough to cause bleeding, although no abnormal indices of coagulation were noted in the present case. In addition, increased platelet fragility resulting from turbulence across the calcified aortic valve may contribute to the bleeding. Interestingly, aortic valve replacement is more likely to cure bleeding in these patients than resection of the compromised bowel which further supports the existence of a relationship between AS and gastrointestinal bleeding. Furthermore, Warkentin et al. recently reported that AS can be complicated by acquired Type II von Willebrand's disease, which is corrected after aortic valve replacement.

In the present case, AS was confirmed both by cardiac catheterization and at autopsy, and the angiodyplasia resembled that described previously. To our knowledge, this is the first case of AS in Japan which has been reported to have exhibited gastrointestinal bleeding from angiodyplasia. Moreover, there have been only a few cases of angiodyplastic lesions throughout the entire gastrointestinal tract, as in this case.

The pathophysiological basis for the gastrointestinal bleeding associated with AS remains unclear. However, we should consider angiodyplasia as a possible cause of

Japanese Circulation Journal Vol. 58, June 1994
bleeding in elderly patients with AS who present with unexplained gastrointestinal bleeding.

REFERENCES