**CASE REPORT**

**Infected endocarditis from Enterococcus faecalis complicating colonoscopy in Heyde’s syndrome**

M Giusti de Marle, A Sgreccia, E Carmenini, S Morelli

A case of infective endocarditis from Enterococcus faecalis after colonoscopy in a patient with aortic stenoinssufficiency and bleeding intestinal angiodysplasia (Heyde’s syndrome) is reported.

A 77 year old man with aortic stenoinssufficiency presented with enterorrhagia and underwent a colonoscopy, which showed normal findings. Fifteen days later he developed a moderate degree of fever. Blood cultures were positive for E faecalis. An echocardiogram showed aortic valve vegetations, and infective endocarditis was diagnosed and successfully treated with antibiotics. Some months later, intestinal bleeding recurred and intestinal resection was performed. Histopathology showed angiodysplasia.

In patients with Heyde’s syndrome antibiotic prophylaxis should be considered before colonoscopy.

Current international guidelines consider colonoscopy to be a low risk procedure for infectious complications.  In fact, published cases of infective endocarditis after proctosigmoidoscopy or colonoscopy are quite uncommon (table 1). Enterococcus is the most frequent causative agent of infective endocarditis complicating proctosigmoidoscopy, particularly in patients with underlying valvular heart lesions (table 1).

We report a case of infective endocarditis from Enterococcus faecalis after colonoscopy in a patient with aortic stenoinssufficiency and bleeding intestinal angiodysplasia (Heyde’s syndrome).  

**DISCUSSION**

E faecalis makes up a significant portion of the normal gut flora of almost all humans, and enterococci account for approximately 5% to 18% of all cases in most series of infective endocarditis. The probability of enterococcal endocarditis increases in the presence of valvular heart abnormalities, gastrointestinal neoplasia, and surgery, including dental manipulation. The most common portals of entry for enterococcal bacteria include the urinary tract, abdominal sepsis, decubitus ulcers, burns, infection of diabetic foot, and

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### Table 1  
Review of reports of infective endocarditis after proctosigmoidoscopy and colonoscopy

<table>
<thead>
<tr>
<th>Reference (publication date)</th>
<th>Endoscopic procedure</th>
<th>Organism</th>
<th>Valvular lesions</th>
</tr>
</thead>
<tbody>
<tr>
<td>2  (1977)</td>
<td>Rigid proctosigmoidoscopy</td>
<td>Enterococcus faecalis</td>
<td>None</td>
</tr>
<tr>
<td>3  (1984)</td>
<td>Rigid proctosigmoidoscopy</td>
<td>Enterococcus*</td>
<td>Mitral prolapse</td>
</tr>
<tr>
<td>5  (1984)</td>
<td>Flexible proctosigmoidoscopy</td>
<td>Enterococcus</td>
<td>Mitral prosthesis</td>
</tr>
<tr>
<td>8  (1988)</td>
<td>Colonoscopy</td>
<td>Streptococcus bovis</td>
<td>Aortic sclerosis</td>
</tr>
<tr>
<td>9  (1991)</td>
<td>Flexible proctosigmoidoscopy</td>
<td>Streptococcus sanguis</td>
<td>AR</td>
</tr>
<tr>
<td>10 (2001)</td>
<td>Colonoscopy</td>
<td>Lactobacillus rhamnosus</td>
<td>None</td>
</tr>
</tbody>
</table>

AR, aortic regurgitation; MR, mitral regurgitation; MS, mitral stenosis. *Not further specified in these studies.
vascular catheters. The gastrointestinal tract has also been reported as portal of entry for enterococci.

In our patient, the probable source for enterococcal bacteraemia was the colonoscopic procedure, whereas both arteriography and upper gastrointestinal endoscopy were very unlikely with regard to this. Indeed, the only endoscopic procedure reported in causal association with enterococcal infective endocarditis is sigmoidoscopy. No cases of enterococcal infective endocarditis after colonoscopy have been described in the literature, as far as we know. On the other hand, three cases of non-enterococcal infective endocarditis have been reported, and only two of them were attributable to colonoscopy. After colonoscopy, the bacteremia rate is low, up to 4%. Thus, antibiotic prophylaxis is not recommended even in patients with an increased risk of endocarditis, as our patient may be considered as being affected by severe valvular heart disease. Bleeding intestinal angiodysplasia could be regarded as an additional risk factor, since bleeding from mucosal damage during endoscopy may increase because of acquired platelet dysfunction in patients with Heyde’s syndrome. Stenotic aortic valve would accelerate the clearance of the largest plasma multimers of von Willebrand’s factor, which mediates the adhesion of platelets to the subendothelium of damaged blood vessels. The reversibility of platelet dysfunction as well as the disappearance of recurrent bleedings after aortic valve replacement strongly suggests a causal link between valvular stenosis and gastrointestinal angiodysplasia.

Conclusions

E faecalis endocarditis may occur on native aortic valve after colonoscopy in patients with Heyde’s syndrome. Thus, appropriate antibiotic prophylaxis should be considered in patients with suspected Heyde’s syndrome undergoing colonoscopy.

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REFERENCES


Summary points

• Heyde’s syndrome is characterised by aortic stenosis and bleeding gastrointestinal angiodysplasia.
• Bleeding gastrointestinal angiodysplasia is caused by deficiency of the largest plasma multimers of von Willebrand factor.
• Aortic valve replacement cures gastrointestinal bleeding and normalises plasma levels of the largest plasma multimers of von Willebrand factor.
• Diagnosis of angiodysplasia is made by colonoscopy and biopsy with corroborating histological documentation.
• Bleeding gastrointestinal angiodysplasia could be regarded as risk factor for enterococcal bacteraemia during colonoscopy.
• Antibiotic prophylaxis should be considered in patients with suspected Heyde’s syndrome undergoing colonoscopy.
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