Angiodysplasia occurring in jejunal diverticulosis

EDWARD A JONES, MD, HUGH CHAUN, BM, FRCP, FRCPC, PHILLIP SWITZER, MD, FRCPC,
DAVID J CLOW, MD, RONALD J HANCOCK, MD, FRCSC

ABSTRACT: The first case of angiodysplasia occurring in acquired jejunal diverticulosis is reported. The patient presented with occult gastrointestinal bleeding and chronic anemia, and was treated successfully by resection of a 25 cm long segment of jejunum. Possible pathogenetic mechanisms for both angiodysplasia and jejunal diverticulosis are discussed. *Can J Gastroenterol* 1990;4(4):151-153

Key Words: Anemia, Angiodysplasia, Jejunal diverticulosis, Occult gastrointestinal bleeding

Angiodysplasie et diverticulose jéjunale

RESUME: On rapporte le premier cas d'angiodysplasie survenant dans une diverticulose jéjunale acquise. Le patient souffrait d'un saignement gastro-intestinal occulte et d'anémie chronique et fut traité avec succès par la résection du segment de jéjunum en cause (25 cm de longueur). Les mécanismes pathogènes possibles et de l'angiodysplasie et de la diverticulose jéjunale sont examinés.

ANGIODYSPLASIA OF THE GASTRO-intestinal tract is an acquired lesion of small submucosal and mucosal blood vessels which can give rise to hemorrhage (1). It is commonly found in the colon but also occurs with less frequency in the small intestine and

stomach (2). Similarly, diverticulosis of the gastrointestinal tract can be an acquired condition which arises with highest frequency in the colon but which may also be found in the small intestine (3). Hemorrhage from such diverticula is a well recognized com-

plication which is considered to be secondary to erosion of normal blood vessels (3). This report describes a case of angiodysplasia which arose in diverticula of acquired jejunal diverticulosis and was the cause of chronic blood loss.

CASE PRESENTATION

A 68-year-old man was admitted to hospital for investigation of iron deficiency anemia. He had presented four years earlier with marked fatigue and occult gastrointestinal bleeding, and was found to have chronic antral gastritis, a large duodenal diverticulum, jejunal diverticulosis and several small benign colonic adenomas. He had no evidence of inflammatory bowel disease or any disease of an immunological nature which might be associated with jejunal diverticulosis. There was no family history of jejunal diverticulosis. His serum B₁₂ was normal. Despite removal of the colonic adenomas, his stools continued to be positive for occult blood, and even though he was treated with iron, his hemoglobin remained about 80 g/L. Physical examination and a labelled red blood cell study for gastrointestinal tract bleeding were normal. A superior mesenteric arteriogram demonstrated an early fill-

Departments of Pathology, Medicine, Radiology and Surgery, University Hospital, University of British Columbia, Vancouver, British Columbia

Correspondence and reprints: Dr Hugh Chaun, Suite 601, 805 West Broadway, Vancouver, British Columbia V5Z 1K1. Telephone (604) 872-0717

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ing jejunal vein and a vascular tuft (Figure 1), features consistent with a diagnosis of angiodysplasia. Previous endoscopic examinations had revealed no angiodysplastic lesions in the large bowel, stomach or duodenum. The

patient had no evidence of aortic stenosis or other cardiac disease. He had not taken nonsteroidal anti-inflammatory drugs or potassium tablets which might have caused mucosal damage and precipitated bleeding of the vascular

lesion. Preoperatively, the superior mesenteric arteriogram was repeated and the catheter left in situ, with the tip lying in the fourth arcade of the jejunal branch artery. At laparotomy before the small bowel was manipulated, a bolus of methylene blue dye was injected into the catheter. A 15 cm segment of mid-jejunum involved by diverticulosis was visibly stained. A 25 cm long segment of jejunum including the stained segment was resected and primary anastomosis performed. The patient has remained well four years later, with a normal hemoglobin.

Examination of the external surface of the resected jejunum showed that more than 20 paramesenteric diverticula up to 1.5 cm in size were present. A mesenteric artery was injected with a warm solution of gelatin and barium, and a specimen x-ray was obtained.

The resected segment of bowel was fixed in 10% formalin. It was sectioned and a careful inspection revealed four (up to 3 mm in diameter) white mucosal lesions in separate diverticula. One of these had a small central depression. The mucosa away from the diverticula was unremarkable. Microscopic examination showed that the lesions consisted of markedly dilated thin walled mucosal vessels (Figure 2) which were continuous with similar submucosal vessels, an appearance diagnostic of angiodysplasia. There was focal erosion of two of these lesions which was the cause of this patient's bleeding (Figure 3).

Figure 1 Later arterial phase of superior mesenteric arteriogram showing early venous filling (left arrow) and vascular tuft (right arrow)

Figure 2 Angiodysplasia in a jejunal diverticulum with ectatic mucosal vessels (top arrows) draining into a submucosal vein (bottom arrow). (Hematoxylin and eosin $\times 35$)

Figure 3 Angiodysplasia in a jejunal diverticulum with eroded mucosa (top arrow) and ectatic blood vessels (bottom arrow). (Hematoxylin and eosin $\times 70$)

DISCUSSION

Jejunal diverticulosis is not a rare condition; prospective autopsy studies using specimen insufflation techniques have documented an incidence of up to 4.6% (4). Although often innocuous, they are a potential cause of malabsorption, pseudo-obstruction, mechanical obstruction, volvulus, perforation, anemia, abscess formation and hemorrhage (3,5). Bleeding from jejunal diverticulosis was first reported by Braithwaite in 1923 (6). Most bleeding from diverticula arises from normal blood vessels. An injury to the mucosa due to ulceration or direct trauma from concretions may breach vascular integrity. In the present case, the hemorrhage arose from angiodysplasia in the diverticula.

Chronic intermittent obstruction of veins as they pass through the muscularis propria has been proposed in the pathogenesis of angiodysplasia of the colon. The increased wall tension that is present in the cecum according to Laplace's principle is the explanation given for the increased prevalence of angiodysplasia in that part of the colon (1). Interestingly, uncoordinated muscular contractions with increased intraluminal pressure have been

considered to be a cause of small bowel diverticulosis (3), and it is logical to believe that such a state could result in angiodysplasia in the jejunum. Indeed there has been one report of angiodysplasia and diverticulosis occurring simultaneously in a segment of the jejunum (7). In that case the angiodysplasia was located in bowel mucosa away from the diverticula.

In the present case the angiodysplasia was present in diverticula, although it is possible that more sensitive techniques may have disclosed evidence also of early angiodysplasia in bowel away from the diverticula. The veins draining these diverticula do not have to pass through the muscularis propria to reach the serosa and therefore cannot be obstructed at this level as has been suggested for angiodysplasia of the colon. Expansion of these thin walled diverticula during periods of small intestinal contractions could possibly cause obstruction of the veins.

To the best of the authors' knowledge this is the first case of angiodysplasia that has been reported to occur in acquired jejunal diverticulosis; therefore, the incidence of this occurrence is unknown. It might possibly be found more often if it was specifically

sought. It is also possible that angiodysplasia might have been responsible for previously reported cases of hemorrhage from jejunal diverticulosis.

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