INTRODUCTION
When conventional diagnostic tests fail to show the cause of gastrointestinal haemorrhage we must bear in mind the possibility of arteriovenous fistulas.
Since 1960 when Margolis et al. (11) showed in angiographic studies that arteriovenous fistulas exist, a number of authors have described angiomatous lesions that may be confused with arteriovenous ones (2, 9).
Consequently, there is a need to describe how to differentiate arteriovenous fistulas from intestinal haemangiomas. Haemangiomas are generally macroscopic lesions that can easily be seen and some cases are detected by barium studies; they are palpable masses found often in the stomach, small intestine, and rectum. Arteriovenous fistulas, on the other hand, are microscopic lesions generally found in the right colon that cannot be detected by palpation or by conventional diagnostic methods.
When Meyer et al. (12) reviewed published reports and documented 22 of their own cases in 1981, they found only 218 cases that involved arteriographic studies.
Our two cases, post-operatively confirmed to be arteriovenous fistulas, serve to indicate not only the effectiveness of laparoscopy to explore and treat this difficult condition, but also the complications that may occur even with minimally invasive surgery in elderly patients with arteriovenous fistulas and several life-threatening diseases.

CASE 1
A 25-year-old woman was admitted with a history of intermittent rectal bleeding for several years with no other gastrointestinal symptoms. Her haemodynamic state was stable, but her mucosa was pale. At digital rectal exploration the finger was covered with red blood. Haematological analysis showed that she had a normocytic, normochromic anaemia. Endoscopy of the upper gastrointestinal tract, intestinal transit study, and colonoscopy were within normal limits. Mesenteric arteriography showed that the terminal branches of the ileocaecal artery were tortuous and abnormally wide (Fig. 1). We decided to treat her laparoscopically.
A Verres needle was introduced into the umbilical region under general anaesthesia, to induce pneumoperitoneum. Three ports were used: a 10 mm one in the umbilicus, another 10 mm one in the left subcostal area in the midclavicular line, and a 5 mm one in the left iliac fossa. We introduced the optic through the umbilical trocar to explore the entire intestine and the abdominal cavity and noted that the segment of ileum that had looked abnormal on the arteriogram was thickened. We exteriorised this loop through a McBurney’s incision, resected the diseased segment, anastomosed the two ends of healthy intestine extracorpore-
ally using a side-to-side technique because they were of different calibres. After replacing the intestine within the peritoneal cavity, and checking for bleeding, we vented the pneumoperitoneum and closed the three trocar incisions with sutures. Postoperative histological examination showed that the submucosal vessels were dilated and that the submucosa was thickened (Fig. 2). The diagnosis was arteriovenous malformation. The patient’s recovery was satisfactory and she reported no further haemorrhagic episodes during the following year.

CASE 2

A 69-year-old woman was admitted with a history of recurrent, self-limiting rectal haemorrhages that had needed repeated blood transfusions. She also had chronic renal insufficiency, she had had a left nephrectomy, was hypertensive, and had ischaemic heart disease. She had had two episodes of upper digestive tract haemorrhage caused by a duodenal ulcer and had had her appendix removed. Endoscopic exploration was not diagnostic, and the nuclear medicine study showed several active haemorrhagic foci around the ileum, in the caecal region, and in the right colon. A selective superior mesenteric arteriogram showed angiodysplasia (Fig. 3). Because of the continued multiple digestive haemorrhages we decided to treat her laparoscopically. Routine blood tests did not contraindicate operation.

Under general anaesthesia with orotracheal intubation we inserted a nasogastric tube and a bladder catheter. After we had introduced a Verres needle in the umbilical region we insufflated the abdomen with carbon dioxide to induce a pneumoperitoneum. We used four ports: one 10 mm trocar in the umbilical region, another in the left hypochondrium, and another in the left iliac fossa, with a 5 mm trocar in the right flank. Once the optic had been introduced we saw a small amount of blood in the abdominal cavity and telangiectasia on the peritoneal surface probably the result of her haematological condition, because we found no other obvious cause of the blood in the abdominal cavity and it was certainly not caused by the laparoscopy. We used electrocautery to mobilise the ascending colon by opening the fascia of Toldt, and examined the retroperitoneal structures. The colon was exteriorised through a 4 cm transverse incision and we carried out an extracorporeal right hemicolecctomy. After the intestine had been replaced within the abdominal cavity, we checked for bleeding, but all seemed well. We placed a Penrose drain in the right paracolic gutter. Her initial postoperative recovery was slow. Intestinal transit was normal by 72 hours postoperatively, and the abdomen was satisfactory. Recovery was complicated by secondary haemorrhages because she had a complex platelet disease caused by a lack of platelet membrane glycoproteins (Ib-IX).

Fig. 2. Photomicrograph of Case 1. Showing many dilated vessels. Haematoxylin and eosin, original magnification (×10).

Fig. 3. Angiogram of Case 2 showing several tortuous vessels and a haemorrhagic area at the level of the ileocolic artery. There is a catheter in the right ureter.
(Bernard-Soulier syndrome) that gave rise to episodes of epistaxis, haemorrhage of the upper digestive tract, haematuria and haemoptysis.

She had acute renal insufficiency that required haemodialysis and she later developed hepatic insufficiency because of viral hepatitis, which had been transmitted by the multiple transfusions. She then developed respiratory insufficiency with associated pneumonia of the right base, and her condition deteriorated until multisystem failure led to her death 45 days postoperatively. The fact that the histological section (Fig. 4) showed sinuous giant submucosal vessels and thickening of the submucosa confirmed our preliminary diagnosis of arteriovenous fistula.

**DISCUSSION**

The aetiology of intestinal arteriovenous malformations is not yet clearly understood. Some authors consider that the phenomenon is congenital (5, 8, 10) and that there is a significant association with aortic stenosis. Meyer et al. (12) identified 18 patients less than 50 years old (mean age, 33 years). In these patients the fistulas were found in the small intestine, the stomach, the rectum, or the pancreas rather than in the right colon. Our first patient had the characteristics of this group.

Other authors consider that these conditions are acquired. Baum et al. (1) suggests that fistulas result from intermittent occlusions that follow episodes of vigorous peristalsis that increase the pressure within the lumen. This excessive pressure produces the arteriovenous fistulas in the submucosa. Boley et al. (4) also considers that the arteriovenous fistulas are acquired, and based their opinion on the fact that these malformations often develop in the ascending colon of elderly people.

The histological sections of both our patients showed vascular lesions. The submucosal vessels were dilated and deformed, and they had thin walls that in some places had only one layer of endothelial cells. Subsequent rupture of these thin-walled vessels gave rise to the bleeds.

According to Boley et al. (3), slow venous emptying is the most notable sign of arteriovenous fistulas, and the next is the convoluted tuft-like profile of the abnormal vessels. Rapid venous filling is seen in only a few cases.

Moore et al. (13) classified patients into three groups according to: their angiographic characteristics, the age of the patient, and the presence of hereditary factors. The first group includes patients over 55 years old in whom the malformation is always found in the caecum and in the right colon. The patients of the second group are younger (mean age 32 years) and their malformations develop in the colon, jejunum, or stomach. Moore et al. reported that during operations on some of his younger patients he found macroscopic evidence of large vessels in the mesentery and in the intestinal serosa. The third group of patients are those whose family histories include several members with hereditary conditions such as Osler-Rendu-Weber syndrome. Many of these patients present with multiple punctate angiomas not only in the intestine, but also in the pancreas, the liver, the buccal mucosa, as well as facial telangiectasia.

Our first patient may be a Moore type II, and the second a type I.

Most of these patients need surgical treatment, and right hemicolecotomy is the treatment of choice for those with fistulas in the caecum. The others will generally need a limited resection of the area that contains the lesion, but it is often difficult to find the lesions (15).

The lesions in both our patients were found at the level of the ileocolic artery. In the first patient we found that the lesion was enclosed within an enlarged and thickened segment of the ileum. However, in the
second patient we found no diagnostic sign. We treated the first patient with a partial resection and the second with a right hemicolecction by assisted laparoscopy. This technique has the undoubted advantage that postoperative recuperation is rapid. Several factors contribute to this; the minilaparotomy is only 4cm long, there is no postoperative ileus, and the patients do not have the postoperative pain that would they would after a laparotomy.

Reported postoperative mortality rates are between 10% and 50% (14, 16). Smith (14) recorded three deaths in a series of 17 patients. His patients, like our second patient, had delayed coagulation associated with abnormalities of platelet function. This high incidence of defective haemostasis suggests that this type of patient is predisposed to haemorrhage and during the prolonged postoperative period new haemorrhagic incidents sometimes occur. The reported incidence of recurrent haemorrhage ranges from 5%–37%.

This high rate of recurrence led Bowers and Dixon (6) to pioneer the treatment of these difficult cases with argon-laser, and Cello and Grendell (7) published a series of 43 patients treated in this way. The technique does not completely eliminate the possibility of further haemorrhage, but it does decrease the need for repeated transfusions. We think that if resection is decided on, then today there is no doubt that this should be by assisted laparoscopy because it eliminates paralytic ileus, greatly reduces postoperative pain, and allows the patients to return quickly to useful life. The fact that laparoscopic surgery greatly reduces surgical trauma is particularly advantageous for those patients who have a coexisting life-threatening diseases like our second patient.

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REFERENCES


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