Three cases of ischaemic colitis

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In recent years there has been growing interest in vascular disease of the intestine. Marston, Pheils, Thomas, and Morson (1966) reviewed 16 cases of ischaemic colitis and described various syndromes included in this condition. I have documented three further cases which, although in general are similar to the above cases, have some particular features of special interest.

CASE 1

E.S. was a woman of 77 years, who was admitted to hospital on 3 May 1965. She had been on Aldomet tablets for hypertension for one year, but was active and fit. For 10 days before admission, she had complained of abdominal pain and had gradually weakened. A few hours before her admission the abdominal pain had increased in severity and her general condition had deteriorated rapidly. During this 10-day period, she had only opened her bowels on one day, when she passed several loose motions but no blood or mucus.

On examination she was found to be extremely ill. She was in peripheral circulatory failure, her blood pressure was 90/60 mm Hg, and her pulse was 90 per minute. She was dehydrated. She had marked abdominal distension with moderate rigidity and tenderness in the lower abdomen. Bowel sounds were absent. On rectal examination, she was tender in the pouch of Douglas. The faecal smear was normal. Her heart was found to be enlarged with left ventricular hypertrophy. Her peripheral pulses were all present.

Some immediate investigations were carried out. Haemoglobin was 102%, total white cell count 13,200 per cu mm (95% polymorphs), sedimentation rate 44 mm/hour, an ECG showed bundle branch block and left ventricular strain, and a chest radiograph showed an enlarged heart.

Following resuscitation, she was taken to the theatre. On the way to the theatre a plain radiograph of the abdomen was taken in the supine position (Fig. 1), and it showed a distended loop of bowel, which was later found to be the transverse colon. This colonic loop had lost its haustrations; this could only be confused with 'toxic dilatation' of the colon in ulcerative colitis. However, the dilatation is so localized that this x-ray picture is almost diagnostic of gangrenous colitis.

At operation the transverse colon was found to be gangrenous. The hepatic and splenic flexures were involved to a lesser extent and the ascending and

descending colons were oedematous. The involved colon was resected; the proximal ascending colon and the sigmoid colon were brought out as terminal colostomies. At operation an attempt was made to feel pulsations in the mesenteric vessels but, as the patient's blood pressure was low and her general condition poor, time could not be allowed for a detailed examination.

The postoperative course was stormy and she died on the ninth postoperative day. During this period she developed renal failure. For the first five postoperative days she passed less than 50 ml of urine each day, the blood urea level rose to 250 mg%, serum potassium to 6 m-equiv/litre, and she became acidotic. The concentration of urea in the urine was less than five times the concentration of urea in the blood. She was treated by limiting the fluid and protein intake, administering carbohydrate intravenously, and using an ion exchange resin. At the time of her death kidney function was

FIG. 1. Plain radiograph of the abdomen (supine).

FIG. 2

FIG. 2. Low-power photomicrograph of colonic wall.

FIG. 3

FIG. 3. Photomicrograph of mesenteric vessels, showing a thrombosed small vein next to a patent artery.

recovering and, at necropsy, no immediate cause of death could be found. There had been no extension of the gangrenous process and no unexpected pathology was demonstrated.

The removed colon was subjected to bacteriological and histological examination. No pathogenic organisms were demonstrated and, in particular, no *Clostridia* were found (Painter, Lee, and Reed, 1966). In the centre of the transverse colon all coats of the bowel wall showed necrosis. In the region of the colonic flexures, the microscopic picture was similar to that described by Morson (Marston *et al.*, 1966). The mucosal layer was more damaged than the outer muscle layers. The submucosa was oedematous and infiltrated by inflammatory cells (Fig. 2). Examination of the mesentery showed venous thrombosis (Fig. 3).

CASE 2

E.G. was a woman aged 92 years, who was admitted to hospital on 20 January 1965. There was no significant past history. During the two weeks before admission she had experienced mild generalized abdominal colic and her abdomen had become distended. Over this period she had passed very little rectally and, in particular, no

blood or mucus. Just before admission she had started to vomit her food.

On examination she appeared well but showed signs of mild dehydration. The abdomen was moderately distended, there was visible peristalsis, and she was tender in the epigastrium. She was normotensive (140/90 mm Hg) but the heart was enlarged. The peripheral pulses were present but the vessels were hardened and tortuous. There was a systolic ejection murmur over the left ventricular outflow tract. A similar but louder murmur was heard in the epigastrium and was transmitted into both femoral arteries.

The peripheral blood picture and electrolytes were normal, an ECG showed left ventricular strain, stool examination showed no pathogens or blood, and a chest radiograph showed an enlarged heart.

The patient was treated as a case of subacute intestinal obstruction. Within 24 hours she had improved and, up to the time of writing, she has remained in very good health with no abdominal or bowel troubles.

Barium studies were carried out following her discharge from hospital. A barium meal and follow through, performed 10 days after admission, showed no abnormality, but visualization of the colon was inadequate. Two barium enemata were performed, one 20 days after

CASE 3

FIG. 4. Barium enema, showing stricture involving the proximal transverse colon.

FIG. 5. Barium enema, showing complete recovery within three months.

admission and the second three months later. The first showed a long stricture involving the proximal transverse colon (Fig. 4); the second showed complete recovery of this portion of the bowel (Fig. 5).

G.R. was a man of 75 years, who was admitted to hospital on 10 May 1966. He gave a past history of malaria and symptoms of generalized arterial disease. For eight years he had experienced claudication in both calves, and, more recently, had suffered from 'night cramps'. For four years he had suffered from attacks of dizziness, occurring once every one to two months. For two years angina had limited his walking to 10 yards.

Two years previously he had suffered a haematemesis. This was investigated as an out-patient but no cause was found. A year later he had an episode of bloody diarrhoea, lasting two weeks. This settled spontaneously.

On this admission he stated that he had had diarrhoea, associated with the passage of fresh blood and mucus, for six weeks. He experienced a feeling of incomplete rectal evacuation and suprapubic pain relieved by defaecation. He had lost 1 stone in weight during this time.

On examination he was pale and extremely weak. He was wasted and dehydrated. The abdomen was scaphoid and soft with no tenderness and no masses. On rectal examination a loose bloody faecal smear was obtained.

He had systolic hypertension (170/70 mm Hg), an enlarged heart and very tortuous peripheral arteries. Ejection systolic murmurs were heard over all the proximal limb arteries, as well as over the praecordium and epigastrium. The leg pulses were absent below the common femoral vessels.

Haemoglobin was 69%, white cell count 15,000 per cu mm (77% polymorphs), sedimentation rate 66 mm/hour, and serum protein 5.6 g%. A chest radiograph confirmed left ventricular hypertrophy and an ECG showed a left ventricular strain pattern. On microscopic examination of the stool red and white blood cells were seen but no pathogens were grown. The barium enema study (Fig. 6) showed ulceration of the left colon with sparing of the lower sigmoid colon and rectum. The irregular margin of the barium column in the colon seemed to be due to thick folds of oedematous mucosa protruding over small mucosal ulcers. This picture produced by mucosal oedema and submucosal haemorrhage is referred to as 'thumb printing' (Boley, Schwartz, Lash, and Sternhill, 1963). The sparing of the distal large bowel was confirmed by sigmoidoscopy, at which minimal reddening of an otherwise normal mucosa was seen.

This patient was treated as a case of ulcerative colitis. During his five weeks in hospital, the bowel symptoms improved but his cardiac condition deteriorated. He finally died of left ventricular failure.

At necropsy, the left colon was examined. It showed a partially healed ulcerative condition. It was very thin in places where previous ulcers had been replaced by fibrous tissue. Some ulcers still remained.

He was found to have severe generalized arterial disease but, despite this, the coeliac axis, the superior mesenteric artery, and the internal iliac arteries were all patent. The distal abdominal aorta and the inferior mesenteric artery were severely diseased. A probe was passed retrogradely up the inferior mesenteric artery and two tight strictures in series were demonstrated (Fig. 7).

FIG. 6. Barium enema.

FIG. 7. Drawing from a post-mortem specimen, showing aorta and a probe passing up the inferior mesenteric artery (IMA). The origins of the coeliac axis and superior mesenteric artery (SMA) are also shown.

One stricture was at the origin of the inferior mesenteric artery and the second stricture was in the main trunk of the artery.

Histological examination of the colonic wall showed similar changes to those found in case 1 (E.S.). The muscle layers appeared relatively normal, there was submucosal oedema containing chronic inflammatory cells, and, in several areas, the mucosa was absent. A search was made for vessels in the colonic mesentery. Initially the vessels appeared normal but, on further examination, occluded vessels were found. It was realized that it was nearly three months since the onset of the disease and, if that was due to an ischaemic episode, an adequate circulation would have developed by the time of the patient's death.

DISCUSSION

E.S. (case 1) was a case of gangrenous ischaemic colitis of the transverse colon, which appeared to be due to primary venous thrombosis. A similar plain x-ray finding in a future patient would strongly suggest this diagnosis. In the early stages of venous infarction, congestion and haemorrhage are usually more marked than in arterial infarction. However, the end result of both causes of infarction is similar and it may be that venous infarction is a commoner cause of a gangrenous colon than is usually recognized.

E.G. (case 2) was probably a case of ischaemic stricture showing a complete spontaneous resolution. The area of the colon involved is similar to the diseased area in the first case. In the cases described by Marston *et al* (1966), the splenic flexure was almost always involved (13 cases out of 16). This case is unusual in that it shows involvement of the proximal transverse colon alone. The stricture shows some of the characteristics of ischaemic colitis, such as thumb printing and sacculation. The most important feature is probably the complete recovery of the colon without any specific treatment. This would seem to rule out other diseases which were considered (tuberculosis or amoebiasis). This feature, together with the age of the patient, would also make the diagnosis of Crohn's disease unlikely.

The diagnosis of G.R. (case 3) lies between ulcerative colitis and ischaemic colitis. The age of onset would be against the former. In a recently published series of ulcerative colitis, only 1.9% of patients experienced the onset of symptoms above the age of 70 (Watts, De Dombal, Watkinson, and Goligher, 1966). These authors found that the natural history in the older age group was different from that in the remaining cases. It is possible that this group included some cases of ischaemic colitis.

The distribution of the ulcerative condition within the colon would favour ischaemic colitis. The rectum

and distal colon were spared and the brunt of the disease process involved mainly the splenic flexure and the proximal descending colon. As previously mentioned, this is the area most commonly involved by ischaemic colitis. Recently Jones (1966) has suggested that the name 'ulcerative colitis' should be changed to 'idiopathic proctocolitis', thus emphasizing that in almost all cases of ulcerative colitis, the rectum is involved.

This patient suffered from generalized severe obliterative arterial disease, which was known to affect the legs, the heart, and the brain. It would seem reasonable that it might affect the intestine as well.

It is well known that the inferior mesenteric artery can be divided without producing ischaemic changes in the left colon. This division occurs routinely during abdominal aortic surgery. The colon is kept alive by the collateral circulation from the internal iliac arteries via the middle rectal arteries, from the coeliac axis, and from the superior mesenteric artery. In a recent series of cases of intestinal angina (Morris, De Bakey, and Bernhard, 1966), two of the three mesenteric vessels were diseased in 80% of cases. In this patient the flow through the inferior mesenteric artery must have been negligible and the diseased collateral arteries presumably could not provide the necessary alternative blood supply.

The histology of the gut wall is non-specific and it

was disappointing not to find more evidence of vascular thrombosis. However, when considering that the diseased colon was healing, the findings were consistent with an ischaemic disease process. Case 3 was probably a case of transient ischaemic colitis and raises the problem of the differential diagnosis of this condition.

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