SIMPLE ULCERS OF THE COLON

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Although not frequently encountered, simple ulcer of the colon has been recognized as a clinical entity since the description of a case by Creuvelhier in his atlas of anatomy and pathology, published in 1835. It was not until 1895, however, that a case was recorded with microscopic confirmation by Morel and Scheyron. The first wide review of the condition was by Quenu and Duval in 1902.

Clinical Picture

The clinical picture is unfortunately not a characteristic one, and diagnosis is made as a rule only when complications have appeared. We were in fact not able to find any case in the literature where a diagnosis had been made before operation or post-mortem. We feel, however, that if the condition is borne in mind, it will be diagnosed and treated much more often. It seems to us that the condition is probably more common than is at present realized, many cases being diagnosed wrongly as other inflammatory lesions or healing spontaneously. It is interesting to note that all the early cases recorded were found at post-mortem and that most of the recent cases were at first regarded as appendicitis, diverticulitis or carcinoma.

Age and sex do not appear to be significant, cases having been recorded in every decade from the 'teens onwards, and with about equal sex distribution. There are no general signs until complications develop, when pyrexia, raised pulse and malaise may appear. Most cases suffer from chronic constipation. Many of the early cases described were, in fact, only stercoral ulcers due to this. Diarrhoea, however, may be seen, but this is probably a secondary manifestation. In some cases in thin patients, the ulcer can actually be palpated and it is in these cases that an accurate diagnosis is most likely (cases 1 and 3 below). Macroscopic bleeding occurs very rarely, but tests for 'occult blood' are much more likely to be of value.

In the majority of cases, patients will present themselves only when complications develop. Of

these, the most frequent by far is an inflammatory spread due to perforation or incipient perforation. As the commonest site is in the caecum, most cases are diagnosed as appendicitis. Most cases in the ascending and proximal transverse colon are diagnosed similarly. Cases occurring in the rest of the colon are usually treated as diverticulitis.

Of the recorded cases, about three-quarters of the proven ones occur in the caecum, most often opposite to the ileocaecal valve. The others are scattered along the rest of the colon with a slight increase at the splenic flexure.

Pathology

The ulcer itself is very similar to a peptic ulcer as seen in the stomach or duodenum; this has caused much speculative and not very logical thinking which has likened the two more than is reasonable. The size varies greatly; from a pin-hole to an inch or more in diameter. The mucous membrane around is quite healthy, but is pulled in to form radiating folds. The actual edge is clean cut, the depth varying. The floor is usually covered in necrotic tissue or granulations if healing is occurring. The base is not indurated like a malignant ulcer, but is more oedematous due to the inflammatory process. There may be ecchymoses in the floor. The shape is regular; round or oval. Not uncommonly, there are several ulcers present. Pigmentation has been described. It probably does not occur in the true simple ulcer but only in stercoral ulcers.

The course varies greatly. At the one extreme healing occurs, at the other a rapid perforation develops. The latter may be followed by a general peritonitis in the cases where the ulcer is on the peritoneal aspect of the bowel. A local inflammatory mass is common, or an abscess may develop. The latter may be intra- or retro-peritoneal and resemble an appendix abscess. Wider spread of the infective process may, on occasions, lead to sub-phrenic and liver abscesses, empyema or distant adenitis or abscess. Haemorrhage is a rare event on a large scale although reported by Wilkie. So also is stenosis due to scarring. Malignant

FIG. 1.—View of a simple ulcer of the colon from mucosal aspect—Case 3.

nant change must be regarded as a theoretical possibility only.

The histological picture is much more constant than the clinical one. The mucosa shows only a simple breach with healthy mucous membrane adjacent to it. It is in the sub-mucous layer however, that the most characteristic changes occur. There is an infiltration of this layer outside the area of the ulcer, with cells of all types; polymorphs, lymphocytes or plasma cells are seen in varying proportions depending on the acuteness of the ulcer. In the deeper layers, a less severe and more localized inflammatory infiltration is seen. There is also usually some thrombosis in vessels, sometimes with peri-vascular fibrosis. When perforation occurs, it is clearcut through all the layers. In the stage just before it actually takes place, the muscle layers show a region of necrotic tissue infiltrated with polymorphs; on the surface of the bowel there is a layer of fibrin. These features are all seen in the photo-micrographs and were described by Morel and Rispaill. The sub-mucous infiltration is very similar to that seen in the rather rare condition of acute suppurative necrotic colitis. A less severe lesion of similar

FIG. 2.—Ulcer with granulation tissue projecting from its base—Case 2.

type is the chronic inflammatory process with acute exacerbations which is produced in the caecum by chronic faecal accumulation.

Aetiology

The cause of this condition is still not known, the theories being almost as numerous as the reported cases. Constipation is the only frequent common symptom and is often indicted. Next is sepsis, and the *Streptococcus*, *Staphylococcus*, *B. Coli*, *B. Proteus* and others have all been blamed in turn, despite the fact that they are all 'normal' bowel inhabitants. Blood-borne sepsis has also been suggested. The macroscopic resemblance to a peptic ulcer has caused them to be compared; many of the cases lie opposite the ileocaecal valve (as a duodenal ulcer often is opposite the pylorus) and the caecal mucosa is more adherent than that of the rest of the colon (as that of the gastric lesser curve). The survival of gastric acid is not likely (even if it is possible to produce ulcers and a change of pH in rats with a deficient diet!). Gastric mucosa has never been found in these ulcers. A vascular cause might be possible as there are sometimes changes in the vessels, but this would make healing rarer than it is even after surgery. Other theories have cited foreign bodies,

FIG. 3.—Chronic inflammatory change in the mucosa with oedema and infiltration in the sub-mucous layer—Case 2.

FIG. 4.—Peritoneal inflammation with fibrin deposit—Case 4.

FIG. 5.—Granulation tissue at the edge of the ulcer which has penetrated all coats—Case 3.

gout, alcohol, disease of the lymphoid tissue, toxæmia from poisons, pathogens or focal sepsis, trauma and lesions of the central nervous system. In connection with the last, several workers produced lesions in rats by injury to the spinal cord (among them Brown Sequard), but no case has ever been recorded in a human with a paraplegia. When all these theories are considered, it seems most likely that the simplest is right and that the process is a local inflammatory and septic one due to a temporary loss of local resistance of the tissues, a local increase of virulence, or both. The remarkable thing is that it does not occur much more often.

Case Reports

Case 1. Male, aged 28 (Negro). For three days before admission, patient had central abdominal pain aggravated by movement. After two days the pain moved to the right iliac fossa. The appetite was poor but there had been no vomiting. Constipated for two days (enema on day prior to admission). Appendicectomy five years before.

On examination the patient was generally fit, temperature 99.8, pulse 88. There was a mobile tender mass 2 in. in diameter beneath the appendicectomy scar.

Laparotomy showed an ulcer on the posterior wall of the caecum at the level of the ileo-caecal valve. Palpated through the bowel wall, it resembled a peptic ulcer. The caecum was re-attached and the wound closed.

The patient has been observed for four years. The mass disappeared in four weeks and has not reappeared. All other investigations have been negative.

Case 2. Female, aged 43. For fourteen days, patient suffered from diarrhoea with anorexia and nausea. On the day before admission she developed severe continuous pain in the right iliac fossa.

On examination, the patient was generally fit, with normal temperature and pulse. There was tenderness in the right hypochondrium above the site of the pain.

Laparotomy showed an abscess 2 in. in diameter in the great omentum 5 in. distal to the hepatic flexure. This arose from an ulcer $\frac{1}{2}$ in. in diameter on the anti-mesenteric border of the bowel. The ulcer closely resembled a peptic ulcer. Histologically, there were the signs described above, together with erosion of several small muscular arteries. The patient made an uneventful recovery after a right hemi-colectomy with end-to-end anastomosis.

Case 3. Male, aged 45. The patient, a highly nervous individual, had suffered from headaches

for fourteen days and had vomited five times during this period. He had otherwise been well. On the day before admission a 'gnawing' pain appeared in the right iliac fossa.

On examination, he was generally well with normal temperature and pulse. A mobile, tender mass, about the size of a walnut could be felt beneath McBurney's Point.

Laparotomy showed an ulcer 1 in. in diameter on the posterior caecal wall level with the ileo-caecal valve. Macroscopically it resembled a peptic ulcer (see illustration). Histology showed a typical simple ulcer as described above. The patient made an uneventful recovery after right hemi-colectomy with end-to-end anastomosis.

Case 4. Female, aged 49. For two days the patient had steady, generalised abdominal pain with anorexia, but no vomiting. On the day before admission, the pain became more severe and moved to the right iliac fossa.

On examination, the patient was ill, temperature 100.2, pulse 110. Tenderness, rigidity and guarding were present in the right iliac fossa.

Laparotomy showed a retro-peritoneal inflammatory mass in the ileo-caecal angle, due to a perforating ulcer $\frac{1}{2}$ in. in diameter just above the ileo-caecal valve. Macroscopically the ulcer resembled a penetrating peptic ulcer. Histology showed a typical simple ulcer with sub-mucosal infiltration and a perforation $\frac{1}{4}$ in. across at the centre. The patient made an uneventful recovery after a hemi-colectomy with end-to-end anastomosis.

We have been told of two other cases when discussing the above at a clinical meeting, but have refrained from including them as details were not complete. In one a perforated ulcer on the anterior wall of the ascending colon of a middle-aged male was treated successfully by simple closure. In the other, an ulcer diagnosed as a carcinoma of the caecum in an elderly woman, perforated while she was awaiting admission and she died. Post-mortem showed a hole in the anterior wall of the caecum.

Differential Diagnosis

Differential diagnosis must always be very difficult in this condition. Most cases will inevitably be discovered at operation. Even then it is unlikely that in many, the diagnosis can be certain. It has been suggested that the bowel be opened in doubtful cases; the healthy state of the mucosa around the ulcer being the criterion of a simple ulcer. More often, however, the complications will overshadow the ulcer itself and mask its appearance. The most important disease to be differentiated is carcinoma of the colon. A simple ulcer can resemble the ulcerative and stenosing

forms and when leakage occurs the differentiation is doubly difficult. A peritonitis from appendicitis, ulcerative colitis or diverticulitis will also resemble the condition. Stercoral ulcer should not be confused. Amoebic ulcers will be detected by discovery of the organisms in the stools. Tuberculous and syphilitic ulcers will, as a rule, be accompanied by signs of the disease elsewhere. In Creuveilhier's atlas, ulcers were described in both caecum and rectum. It seems likely, however, that these cases were stercoral and syphilitic ulcers respectively. Thus, as not often happens, he established the identity of a disease without actually seeing a case.

Treatment

Opinions on the best treatment vary. It will obviously be affected by whether complications have developed as then they will take prior place. Subject to this, however, it is best to aim at complete extirpation. As the condition can rarely be differentiated from carcinoma with complete certainty, it is advisable to resect as though this were actually present. Less often, local excision is safe. Perforations heal quite well with simple closure. In retrospect, we do not feel that our

treatment of Case 1 was ideal, as, although events showed a satisfactory outcome, the risks we were taking were probably greater than that of resection.

Literature

The literature on this condition has not, on the whole, proved very rewarding. Cameron gives a long and almost complete list. Like him, we found most of the case reports very incomplete and unreliable. Many were not made first-hand and a check of the original reports showed that the case was one of ulcerative colitis, perforation, obstruction, or other lesion. Papers from the nineteenth century are especially poor, but an excellent review was made in 1902 by Quenu and Duval and should be read before consulting the earlier papers. A number of cases have been described by Moore since Cameron's paper was published.

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