Ischaemic colitis*

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The concept of ischaemia as a cause of colitis is a relatively new one (Marston et al., 1966). It seems to be an uncommon type of colitis and is certainly much less frequent than ulcerative colitis and segmental colitis due to Crohn's disease. However, it is possible that in large general hospitals, dealing particularly with geriatric patients, ischaemic colitis may be recognized more frequently in the future. It is a disease that characteristically presents in patients past the age of 50 with a sudden onset of bleeding per rectum and abdominal pain. Ischaemic colitis also occurs in patients after reconstructive operations on the aorta, in diabetics and in association with certain systemic diseases which have a vascular component, such as scleroderma and rheumatoid arthritis.

Ischaemic colitis is usually a segmental inflammation, that is, one in which there is normal bowel on either side of a diseased area. These segments are usually short but may be quite long. They particularly affect the splenic flexure of the colon.

The pathology of ischaemic colitis can be described in three phases. Firstly, the acute phase; this is variously called acute ischaemic colitis or infarction of the colon. So-called necrotizing colitis is probably a form of acute ischaemic disease. Secondly, transient ischaemia of the colon. Lastly, ischaemic stricture.

Before describing these three phases I would like to emphasize that ischaemia may affect the small intestine in just the same form as it affects the colon. Indeed, lesions may be present in both parts of the intestine in the same patient. In the small bowel ischaemic strictures must be distinguished from Crohn's disease. So-called solitary ulcers of the small intestine which seem to have the ingestion of potassium chloride tablets as an initiating factor also have an ischaemic component in their pathogenesis.

Acute ischaemic colitis presents as infarction of the bowel but often the pathological appearances are modified by secondary infection with mucosal ulceration, gangrene and perforation.

Histologically, there is necrosis of tissue, particularly the mucosa, but the muscularis propria remains relatively intact because it is more resistant to acute deprivation of blood. The submucosa is oedematous and here it is common to see areas of haemorrhage and small veins filled with platelet thrombus. Acute ischaemic colitis may affect any part of the colon, but is usually centred on the splenic flexure. In some cases large numbers of Clostridium welchii may be found in the necrotic tissues of the bowel wall, particularly the mucosa and submucosa. These organisms are commonly present in the faeces of normal persons. They will invade the bowel wall if it becomes necrotic.

Transient ischaemia of the colon is not a condition which normally requires surgical treatment and thus specimens for study by the pathologist are not generally available. However, a study of patients with transient ischaemia suggests that the morphological changes which do undoubtedly occur and can be demonstrated radiologically are insufficient to produce any permanent distortion of the tissues of the bowel wall leading to stricture formation.

The pathology of ischaemic strictures of the colon has been my special interest. These have shown a varying degree and extent of stricture formation with mucosal ulceration. Mostly they have been rather short lesions (up to 15 cm long) in the region of the splenic flexure and descending part of the colon. They are uncommon on the right side of the colon and none has involved the rectum. In a few cases the stricture has been long, affecting most of the transverse colon within the area supplied by the middle colic artery. The short strictures have a rather fusiform appearance tapering off gradually at both ends. In others there is a striking irregular sacculcation of the gut wall. The long strictures were tubular with considerable thickening of the bowel wall and narrowing of the lumen. In all cases the submucosa was widened and filled with white granulation tissue. Mucosal ulceration tends to be patchy.

The principal histological features of ischaemic stricture are full-thickness loss of mucosa in the

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ulcerated areas, the surface of which is covered by granulation tissue packed with dilated capillary blood vessels. At the edge of ulcerated areas epithelial regeneration is present with columnar epithelium beginning to grow over the surface. Neighbouring intact mucosa is not always normal but shows patchy atrophy and irregularity of structure suggestive of a healed state with incomplete recovery. The muscularis mucosae adjacent to ulcerated areas appears damaged with spaying of its fibres and fibrosis. The submucosal layer shows the most conspicuous changes, being widened and filled with a characteristic granulation tissue. This consists of marked proliferation of fibroblasts, some oedema and a sprinkling of chronic inflammatory cells, including lymphocytes, eosinophils and plasma cells. There is also an increase in collagen. Many macrophages full of pigment, giving the staining reaction for haemosiderin, are a prominent feature of the cellular infiltrate. This observation is probably of considerable diagnostic importance as identical cells are a feature of the granulation tissue of the resolving inflammation in ischaemic myocarditis. The submucosal arterioles tend to be thick-walled and tortuous. In occasional cases there is prominent fibrinoid necrosis in the wall of submucosal vessels near the surface of ulcerated bowel but this is probably a secondary arteritis. The muscularis propria shows patchy fibrosis with replacement and separation of its fibres by granulation tissue identical and continuous with that seen in the submucosal layer. The inflammatory process also involves the serosa and pericolic tissues, but this is always patchy.

The whole histological picture of this stage of ischaemic colitis strongly resembles the appearances in the resolving stage of ischaemic myocarditis (Mallory, White & Salcedo-Salgar, 1939; Lodge-Patch, 1951) and is distinct from the microscopic pathology of idiopathic proctocolitis or Crohn's disease. Transmural hyperplasia of lymphadenoid tissue, intramural fissuring and sarcoid granulomata are conspicuously absent. Crypt abscesses are only occasionally seen and inflammatory polyposis is not a feature. Moreover, haemosiderin-laden macrophages have not been noted in the inflammatory infiltrate of proctocolitis.

The pathogenesis of ischaemic stricture is not always clear. A blocked or absent mesenteric vessel may be demonstrated and this can be due to embolus. Atheromatous embolism to the intestine has been described. The age incidence of ischaemic colitis is of course consistent with that expected in degenerative vascular disease of any organ. Most of our cases have occurred at the splenic flexure which is the point where the superior and inferior mesenteric systems anastomose. It has been shown that the blood supply at this point is precarious (Griffiths, 1956).

If for any reason there is a sudden or prolonged failure of blood supply without a vascular block then a histological infarct may occur. Atheromatous plaques at the origin of the mesenteric vessels are common and small fluctuations in blood pressure across such lesions can result in a reflex shut-down of peripheral vessels leading to bowel necrosis (Marston, 1962). Other factors which may be involved in the pathogenesis of ischaemic colitis are congenital abnormalities of main vessels, vascular spasm, rheumatoid arthritis, scleroderma and diabetes. Polyarteritis nodosa is, of course, a condition which may cause a segmental lesion of the colon, but this is usually an obvious diagnosis histologically.

Ischaemic colitis has been produced in dogs (de Villiers, 1966) and it is interesting that the histology of the lesions closely resembles what is seen in the human disease.

References


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