Leading article

Intestinal ischaemia

Every gastroenterologist, physician or surgeon, in the course of his day-to-day practice sees hundreds of patients with peptic ulcer, gall stones, diverticular disease, spastic colon, and many other complaints, but will come across only one or two in whom the abdominal symptoms are caused by intestinal ischaemia. It is therefore unrealistic to expect even a very busy gastrointestinal specialist to have an extensive experience of this disorder, important though it may be, and we depend on the one or two individuals who make this an especial interest to come forward with a meaningful review of a reasonable series of cases, and to give an authoritative view based on an analysis of direct experience. Hopefully, this approach should give us guidance on diagnosis, selection of appropriate patients for operation, and an assessment of results.

The review in this issue of *Gut* (p. 656) by Mr Adrian Marston is therefore welcomed and it is clear that several important messages emerge from it. The first is how very uncommon it is as a clinical entity. At the Middlesex Hospital there is a centre well known for its special interest in the disorder and yet from the many referrals during a period of 10 years, only 28 were found to have clear evidence that their disability was really due to chronic intestinal ischaemia. It is not too surprising that 20 of these 28 patients had evidence of atherosclerosis affecting other parts of the body. This points to this degenerative arterial disease being the most frequent cause of the ischaemia. When we consider, however, the thousands of operations which are required for atherosclerotic disease affecting the legs, heart and brain and consider the high incidence of narrowing of the main intestinal vessels from both extensive autopsy and aortographic reviews referred to in Mr Marston's article, this once again emphasises that although varying degrees of occlusion are indeed common, the number of patients who develop symptoms which need treatment is very small.

The next lesson is how far from clear cut and uniform are the symptoms of this condition. Because the clinical presentation of intermittent claudication or cardiac angina is usually straightforward, it is natural to assume that patients with intestinal ischaemia should complain of abdominal pain made worse after the muscular exercise induced by eating, and relieved when the meal passes beyond the affected segment. This, however, appears to be quite exceptional, and the commonest clinical syndrome comprises varying combinations of non-specific abdominal pain, bowel disturbance, and weight loss. It is an interesting observation that this latter is more likely to be because of a decreased food intake, than to any marked defect in the processes of digestion or absorption.

Although there are clearly difficulties in measuring with precision by methods available at present either total splanchnic blood flow, or that to

localised segments of the intestine, it is frustrating that the observations which have been made show no clear correlation between the degrees of narrowing of the vessels, symptoms, or indicators of altered intestinal function. In spite of the great difficulties it is nonetheless important to learn more about the natural history of the disorder, and to develop methods of more exact evaluation, for we are faced with two important clinical questions. First, as it is evident that many people do have partial narrowing of their major intestinal arteries, what is the risk in these individuals of the block eventually becoming complete with the risk of acute infarction, and its serious consequences? Can we confidently identify from this large population those who are particularly at risk, and, if so, is it appropriate to offer them some form of prophylactic revascularisation operation? Second, is there a specific pattern of chronic narrowing of the intestinal vessels which is more likely than others to lead to the presentation of abdominal pain, bowel disturbance and weight loss, and if these can be identified, how useful and safe is it to recommend vascular reconstruction to them? Do these operations improve the quality of life and furthermore, does the benefit endure?

It would seem that the first main clinical question is virtually impossible to evaluate in the ideal way of gathering together two comparable groups of patients and randomly allocating them to one standard form of treatment, or no treatment in a properly controlled trial. With so few patients available, a variable picture, and the length of time needed to build up sufficient numbers, such an approach seems quite impracticable. We know that most of those with anatomically detectable narrowing lesions in their intestinal vessels will not be troubled by these during their lives, so that unless we have some method of confidently identifying solely those at risk, it is difficult to see how such a controlled evaluation is ever likely to be achieved.

It is also difficult to see the way ahead with respect to the second main clinical question. After all, the gastrointestinal tract is not simply a muscular organ, the function of which can be directly assessed by measuring and testing the efficiency of this tissue. It is rather the function of the complicated active biochemical machinery within the mucosa which is most important, and although disordered muscle activity may influence the digestive and absorption processes, the two are not necessarily directly correlated in a quantitative manner. It is not surprising, therefore, that there is no good correlation between the incidence and severity of symptoms, the amount of narrowing of the various arteries, and the indices of either total flow to the intestine, or of digestive function. Clearly a considerable overall reduction in blood flow to the intestine can occur without disturbance of the basal function of the gut mucosa. Furthermore, tests which look for alterations in various indices of digestive function after the 'exercise' of a meal, are not strictly analogous to exercise tests to show cardiac angina, or intermittent claudication, because any pain induced by the meal would be attributable to relative ischaemia of the muscle tissue, while the 'function' measurement would relate to mucosal activity.

Unfortunately, recourse to animal experiments does not seem helpful with these vexed clinical questions. While there is no difficulty in producing a range of structural and functional disturbances, there are considerable interspecies variations in response, and of course it is not

possible to evaluate symptoms of a chronic ischaemic nature. Animal models of intestinal ischaemia mostly involve the application of local stricture, or complete occlusion of segments of otherwise healthy vessels, different from the more irregular distribution of blood vessel changes seen in human atherosclerosis.

So for the present the diagnosis and management of chronic intestinal ischaemia remain matters mainly of clinical judgement. When all other possible causes of symptoms have been painstakingly excluded and the possibility of intestinal ischaemia considered, it is reasonable to proceed to aortography with relevant lateral views. If one or more narrowings or occlusions of the main intestinal arteries are shown, the various tests of intestinal function do not seem to help much in the further identification of those who would be most likely to benefit from operation, and those who would not.

It seems, therefore, for the moment that real progress can only be expected from further thorough assessments and reviews of collected series from specialised referral centres. It seems sensible for those encountering patients in whom there is a strong suspicion of such a disorder to refer them to such centres, rather than yielding to the temptation to treat them themselves, so that a sufficiently large body of experience can be brought together for uniform careful analysis and follow up.

One attractive suggestion emerging from the Middlesex study is that patients with chronic intestinal ischaemia might be placed in four separate categories representing increasing grades of severity. This might well help in the more accurate assessment of (a) the natural history of the disorder, (b) the identification of those most at risk, (c) the selection of those most appropriate for surgical treatment, and (d) the results of treatment.

As the extent and distribution of the occluding lesions clearly varies from patient to patient, it is understandable that the actual method of reconstruction has varied, as judged most appropriate to the individual case. Procedures in the Middlesex series varied from simple division of a compressing median arcuate band to angioplasty, reimplantation of the superior mesenteric artery, side-to-side anastomosis of the superior mesenteric artery to the aorta, and retrograde revascularisation from aorta or iliac vessels. We have to accept Mr Marston's conclusion that it is pointless to repeat the various tests of intestinal function after operation. So how are we to assess good and bad results? Must it remain based solely on the patients' accounts of relief of symptoms, for it might be argued that that is really all that matters, providing the follow up period is long enough. Because we haven't any easy and reliable method of measuring total, or muscle, or mucosal blood flow before and after the procedure, would simple pre- and postoperative measurements of intra-arterial pressure not give at least some crude overall guide?

In spite of all the unanswered questions which remain, it is timely and salutary to have this thoughtful and very honest review from a leading authority on this important subject.

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