Abdominal Angina

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Currently specialization in medicine leaves a gap between the special interests of several specialties: internist, gastroenterologist, cardiologist, general abdominal surgeon, and vascular surgeon, into which the problems of the patient with abdominal angina are likely to fall. Although abdominal angina from intestinal ischaemia is an infrequent event, it is lethal unless recognized and corrected by vascular procedures that have been developing. The syndrome deserves more special attention by the practicing physician than it has received during the past half century since I first drew attention to the syndrome [1].

Abdominal angina results from the accumulation of metabolites when inadequate volumes of blood flow through the muscle of the bowel wall to clear them and shares a similar mechanism to that of angina pectoris [2] (Myocardium) and intermittent claudication (leg muscles).

Blood flowing through the 3 main vessels supplying the bowel becomes sufficiently impeded to produce the ischemic pain of abdominal angina; but yet is adequate to prevent necrosis. Available collateral circulation is small, particularly in the distribution of the superior mesenteric artery. The superior mesenteric artery communicates above and around the head of the pancreas, with the celiac axis through the pancreaticoduodenal arteries, and below in the mesentery of the colon in Riolan's arch, where the middle colic branch of the superior mesenteric artery communicates with the left colic branch of the inferior mesenteric artery.

The mesenteric vessels respond to neurogenic, hormonal, and pharmacologic influences, and this response influences the volume of available collateral circulation.

Characteristic Features of the Pain of Abdominal Angina

The 7 characteristics of the patient's pain provide the most valuable clues for recognition of abdominal angina. These are the same characteristics appropriate for recognizing angina pectoris, but have substantially different features for abdominal angina.

1. Location of the pain is poorly localized around the umbilicus or in the epigastrium.
2. Radiation may occur at back but radiation is frequently absent.
3. Quality of the pain varies from a dull ache in some patients to a colicky pain in others. Lack of associated tenderness is characteristic but is present in most other painful conditions.
4. Intensity is usually sufficient severe to discourage eating and to lead to severe loss of weight. It is greater than one might expect with the limited physical findings.
5. Duration varies from few minutes to an hour or more and correlates with intestinal function. It gradually increases, reaches a plateau, and then decreases several hours after eating.
6. Fluctuation and periodicity are characteristic and pain-free intervals separate the attacks that are ordinarily correlated with eating and lead to food avoidance behavior and weight loss.
7. Circumstances surrounding the occurrence and subsidence of the pain are correlated with intestinal function. Ordinarily, it begins 15-30 minutes after meals, but may be delayed by as much as 2 hours if gastric emptying is delayed. The patient often prefers hunger to the pain, eats infrequently in small amounts (the "small meal" syndrome [3]). Mileu not invariably present: atherosclerosis elsewhere, weight loss, assuming some relief from prone or squatting position, sitophobia, fear of eating, functional bowel disturbance with fatty stools.

Physical Examination

Physical examination yields little evidence of abdominal angina and serves mainly to exclude other diseases that could be responsible for abdominal pain. Although evidence of arteriosclerosis elsewhere and a short early systolic bruit may be audible in the periumbilical region, it is frequently absent. Lack of tenderness is characteristic of intestinal angina, while tenderness is expected in most of the other painful diseases of the abdomen.

Clinical laboratory findings

The clinical laboratory examination serves to exclude other diseases.

Radiography

Routine radiographs without contrast material may show small bowel distension and are non-diagnostic.

Angiography is necessary for definitive diagnosis.

Treatment

When I drew attention to abdominal angina, a half century ago and said that it was almost "invariably fatal" unless "cured by appropriate vascular correction," there was little immediate success in the interval since until recently. A few medical procedures: acupuncture, papaverine, anticoagulants, and spinal cord stimulation have been tried with virtually no success. Corrective vascular procedures are the only real hope for curing intestinal angina because it is unusual for the collateral circulation to develop more rapidly than the main blood vessels progressively narrow. Revascularization procedures have shown moderate success and have been reported from individual institutions in small numbers. Although high-grade visceral arteries stenose in patients with intestinal ischemia, symptoms can be treated by either surgical procedures or percutaneous transluminal angioplasty, surgical

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revascularization met with the earlier reported and greatest success, combined with moderate risk.

In 1997, the section on vascular surgery from the department of Surgery of the University of Chicago [4] reported the results of 24 consecutive patients treated in the decade before 1996 with successful outcome of mesenteric bypass with minimal mortality and symptom-free survival rate of 70%, indicating to the presenters that revascularization is the treatment of choice for chronic mesenteric ischemia. In 2002, Pietura et al. [5] from the University of Lubin, in Poland described experiences between 1996 and 2011 with 6 patients with abdominal angina. A good clinical and ultrasound outcome of PTA (perecutaenoustransluminal angioplasty) was achieved, although one patient required implantation of the superior mesenteric artery. In 2003, Jayaprakash Sreenarasinhaiah [6] from the University of Texas analyzed previous experience from throughout the world and concluded that angiographic therapy is a feasible alternative for patients with abdominal angina in whom surgery is considered precarious, but that for others, the mainstay is surgery. He added that medical therapy is mainly supportive. In 2012, Scharafuddin et al. [7] from the University of Iowa analyzed experience between 2004 and 2011, with 27 nonembolic total occlusions of the superior mesenteric or celiac arteries by endovascular recanalization. The procedure proved both feasible and successful provided careful planning was used. Four delayed deaths occurred during the follow-up period and clinical recurrences occurred in 6 patients.

Let us improve recognition to provide the opportunity for successful management.

References