GI

MEDICAL GRAND ROUNDS

Parkland Memorial Hospital

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ISCHEMIC BOWEL DISEASE

In the past, ischemic bowel disease was generally considered to be uncommon, but a fatal event in majority of the patients. Recent estimates, however, suggest that this disease may not be uncommon, as it results in around 2,000 deaths a year in the United States. Many patients who survived in the past did so only after massive bowel resections, which left them with multiple problems associated with short bowel syndrome. In recent years there has been an increasing appreciation that the cases of fatal bowel ischemia represent only a small proportion of cases of ischemic bowel disease. Moreover, it appears that recognition of early forms of ischemia with proper corrective treatment may lead to considerable reduction in the mortality and morbidity in these patients.

The purpose of this presentation is to give you an overview of the clinical spectrum of the ischemic bowel disease and to outline the current views on the methods of diagnosis and proper management of these patients, using examples from my review of 57 proven cases of bowel infarction seen at Parkland Memorial Hospital in the last 4 years.

In order to better appreciate the clinical syndromes of bowel ischemia, it may be helpful to briefly review in general terms the consequences and the causes of bowel ischemia.

Consequences of Ischemia of the Gut

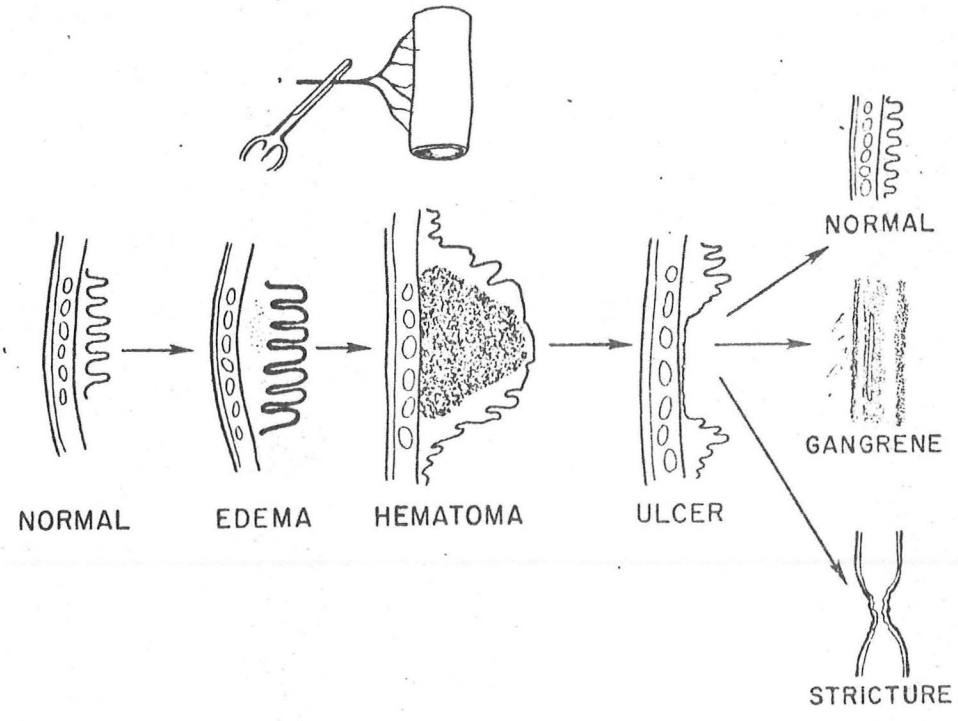
The end result of all forms of ischemia of the gut is death of bowel wall, but it is very intriguing that with acute occlusion of mesenteric circulation "death of the individual frequently occurs before the intestine loses all apparent viability". Peristalsis may sometimes be evoked after death. Furthermore, "release of the experimentally occluded superior mesenteric artery leads to death more quickly and certainly than if the occlusion is maintained".

Bowel changes in ischemia:

The degree (transmural) of the involvement of the ischemic segment of the bowel and the extent of the lesion along the intestine varies with the cause and the degree, duration and rapidity of the ischemic process. Occlusion of different vessels leads to involvement of different segments of the bowel, but the development of the lesion is quite similar in all segments.

When mesenteric artery is suddently occluded, all pulsations disappear and the bowel becomes blue-white and spastic with collapsed arteries and prominent veins. Contractions in the gut increase initially. (This explains increased bowel sound and gasless abdomen in early stages of ischemia.) The contractions, then decrease and eventually disappear, and after several hours bowel loops become relaxed and distended (account for abdominal distension, reduced bowel sounds and x-ray picture of ileus). In complete and rapid ischemic necrosis, all the layers of the bowel wall are "converted into the shadow of their former selves". In most of the cases, 8-10 hrs after the onset edema and hemorrhage appear in the submucosa, and the overlying mucosa undergoes ischemic necrosis. The submucosal edema causes thickening of the mucosal folds. In more severe cases,

BOWEL CHANGES IN GUT ISCHEMIA



the muscle wall is also involved in transmural hemorrhagic infarction. The dead bowel may by invaded by bacteria (particularly E. coli, fecal streptococcus and clostridia). Frank perforation is rare, but peritoneal reaction always occurs and turbid bloody fluid collects in the peritoneum. Occasionally bubbles of gas may appear in the bowel wall and/or the portal vein.

In the less severe cases, the deep muscle layers are relatively well preserved. The submucosal hemorrhages enlarge and may produce luminal occlusion (these changes are responsible for thickness of bowel wall and so-called thumbprints and sometimes picture of bowel obstruction). In milder cases, the hemorrhage is resorbed in 1-2 weeks, and the changes revert to normal. More severe ishemia leads to ulceration of the overlying mucosa at certain areas and also inflammatory reaction. These lesions, when in colon, are grossly quite indistinguishable for ulcerative colitis or Crohn's disease. However, in most of these cases sloughing eventually occurs leading to complete recovery in 1-6 months. In some cases inflammatory reaction predominates and fibrous stricture may form. These may cause bowel obstruction and may require surgery. In some cases, toxic dilatation of bowel may also occur.

The histology of the ischemic lesions of the bowel is very interesting, but will not be discussed here. These changes have been reviewed by in many excellent papers. Few electronmicroscopic studies of mucosa of bowel in low flow states and milder forms of ischemia are also available. (See Ref. 4, 5, 8)

Bowel ischemia is associated with a variety of functional changes in the bowel. The mucosal cells lose their ability to produce mucus, become subject to digestion by intraluminal enzyme, and also become permeable to bacteria and other toxic substances. The resultant cellular dysfunction may lead to loss of fluid and electrolytes, and of plasma into the lumen of the bowel. These losses may be massive and produce marked alterations in fluid and electrolyte balance in the body. Loss of protein may produce a state of protein-losing enteropathy.

Chronic ischemia may lead to malabsorption of a variety of nutrients and may present as malabsorption syndrome.

Systemic effects of bowel ischemia:

Abolition of flow in the mesenteric circuit creates a profoundly complex physiologic situation, which is largely due to three factors, namely: redistribution of body fluids, absorption or release of toxic materials into circulation and reflex mechanisms. Many abnormalities arise from several of these and other undefined mechanisms. Some of these disturbances are:

1) Hemodynamic alterations: Effects on the heart: (cardiac output, coronary blood flow): Several observers have shown that acute mesenteric ischemia is associated with early and significant decrease in cardiac output and coronary blood flow, which manifests by frequent occurrence of subendocardial injury or ischemia on the EKG. Vyden closely followed 13 patients with acute SMA insuffiency. He found that 8 (62%) of these sustained myocardial infarction after the commencement of abdominal catastrophe. He also pointed out that because of abdominal symptoms and other problems, fresh cardiac infarction in these patients may be overlooked. Other workers have shown that mesenteric ischemia in the dogs causes EKG changes which are consistent with left ventricular injury,

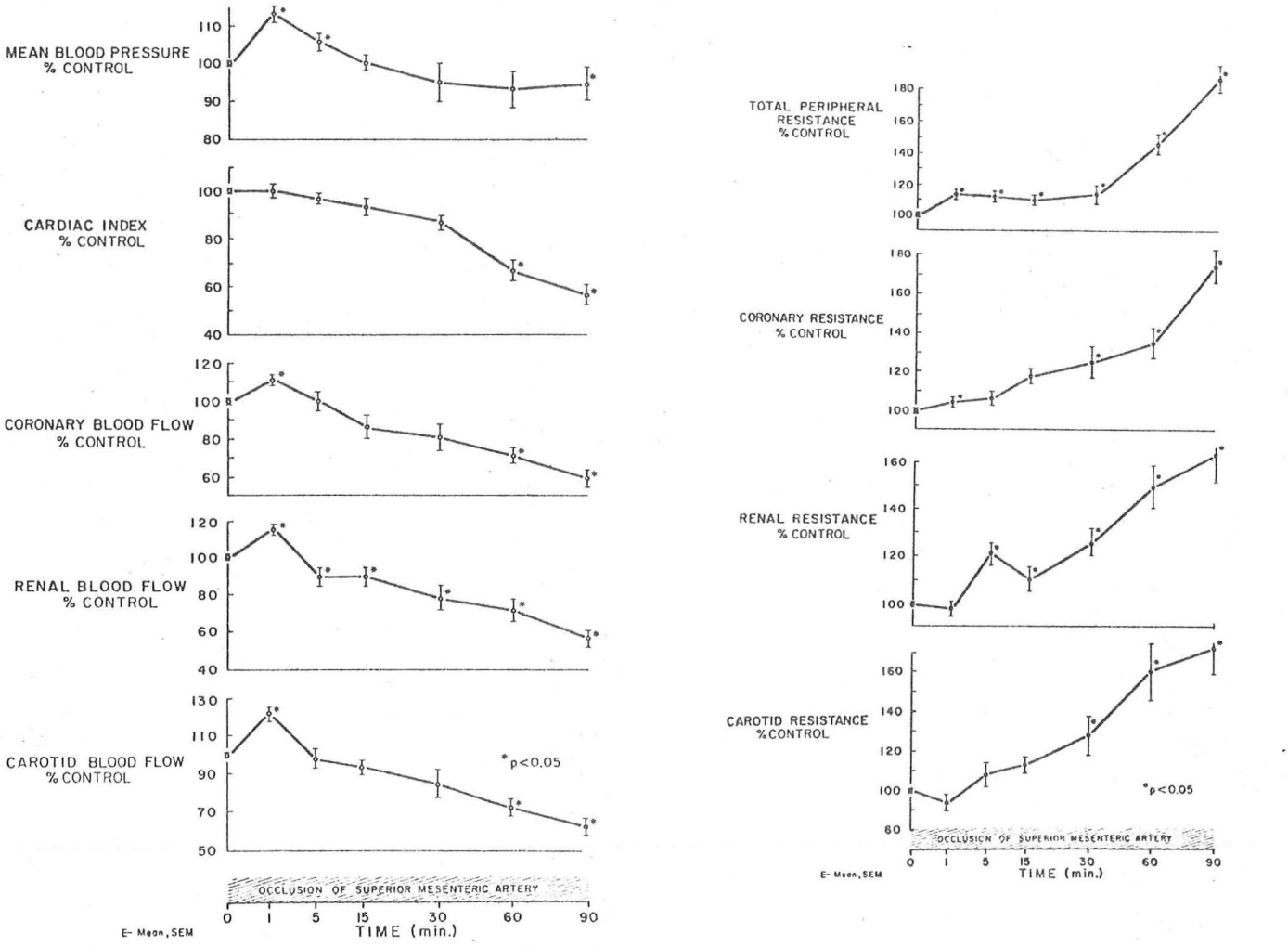
subendocardial ischemia and even infarction. These changes are due to marked reduction in coronary blood flow and increase in peripheral resistance.

The cause of drop in the coronary flow is not hypovolumia or intravascular thrombosis, as the reduction in coronary flow occurs very early before these changes become significant.

A variety of reflex and humoral factors have been incriminated. Lefler and Glenn have suggested a myocardial depressant factor is released in the circulation in splanchnic ischemia. These matters have been reviewed. (See Refs. 11, 14-16)

Arterial blood pressure: Measurement of arterial blood pressure does not adequately reflect the hypoperfusion state in these patients. SMA occlusion causes an initial small but significant increase in the systemic blood pressure. The blood pressure then returns to control levels at which it remains for some hours.

Early hemodynamic effects of acute SMA occlusion in 15 dogs have been summarized in Figure below:



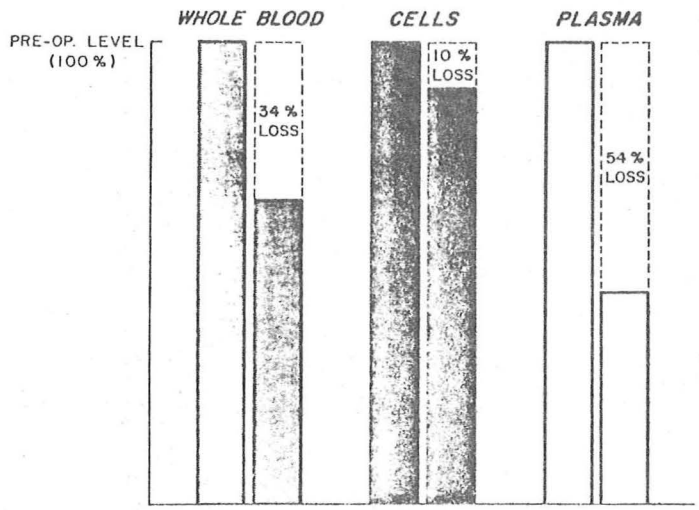
(Vyden, 1971)

2) Volume deficits: With infarction, the capillaries become highly permeable and plasma from any blood that still flows to the bowel is lost into the tissue and the lumen of the gut. Because of the relatively enormous surface area of the small bowel (equivalent, in fact, to the area of the body surface), it is hardly surprising that the effect would be similar to that of a major burn. Hemoconcentration occurs and shock-like state is aggravated.

Although fluid loss continues at a fairly constant rate while the artery is occluded, this rate is sharply increased if ligature is released. In contrast to the occlusion period, which results in plasma leakage, post-release period is characterized by intraluminal hemorrhage, presumably because of exposure of damaged minute vessels of mesenteric bed for arterial pressures.

Mean blood volume losses in 20 dogs following SMA ligation until death are shown in Figure below:

MEAN BLOOD VOLUME LOSSES IN 20 DOGS
FOLLOWING SMA OCCLUSION

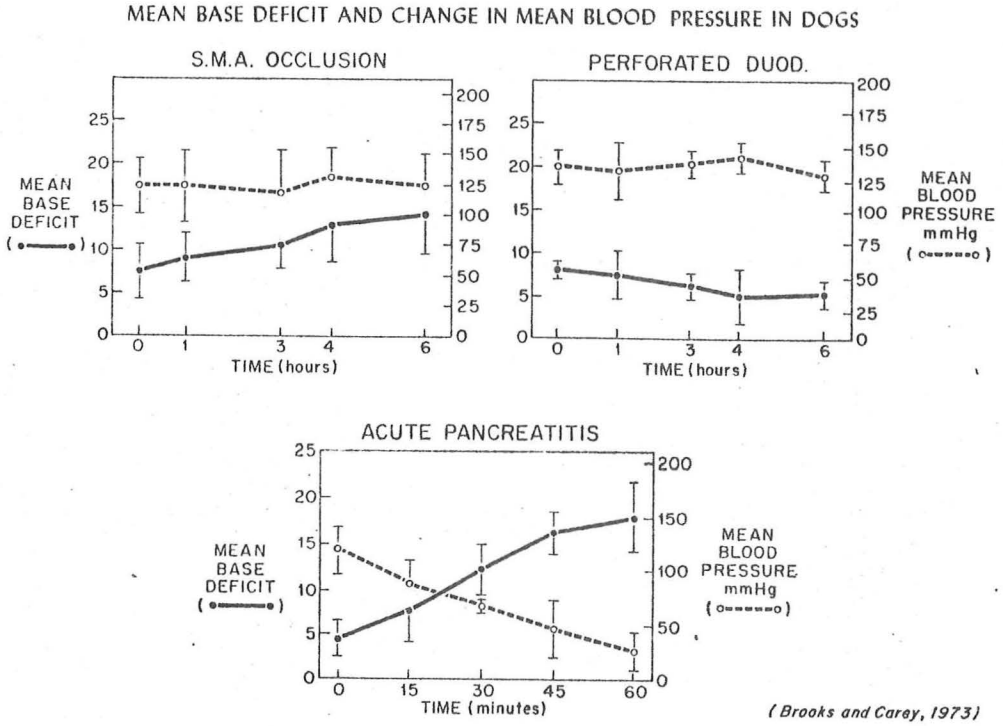


(Marston, 1971)

3) Biochemical alterations: Several alterations in the presence of mesenteric ischemia have been described, but none of these are specific enough to be of real help in diagnosis. However, some of the abnormalities listed below may be of some help.

Metabolic acidosis: Brooks and Carey (1973) estimated the base deficits by using the Siggard-Anderson nomogram (Scand. J. Clin. Lab. Invest. 15:211, 1963) in dogs with SMA occlusion, induced pancreatitis and perforation of duodenum. The results showed that SMA occlusions caused marked base deficits, even when the blood pressure is fairly well maintained. This was quite distinct from the

responses in pancreatitis or duodenal perforation, as shown below:



Hypoamylasemia → Hyperamylasemia: Hypoamylasemia occurs at the onset of acute mesenteric vascular insufficiency, and it has been attributed to marked sequestration of the enzyme with the plasma lost in the bowel and the peritoneal cavity. As the period of gut ischemia lengthens, the amylase levels go up because of the return of the banked up enzyme into the circulation. This biphasic behavior of serum amylase may help to distinguish it from pancreatitis.

Serum protein changes: Due to increased permeability of the anoxic vessels in the gut, there is reduction in serum albumin and also gamma globulin with elevated alpha-1 and alpha-2 globulin and normal beta globulin.

Hypoalbuminemia is responsible for low serum calcium levels sometimes observed in these patients.

Hypogammaglobulinemia is characteristic and interesting in the presence of marked neutrophil leukocytosis. In persistent gut ischemia the gamma globulin increases above normal levels.

Other changes:

Hypocalcemia (Amer. J. Dis. Child. 117:599, 1969)

Alkaline phosphatase elevation (Gut 10:121, 1969)

Alterations in LDH, SGOT, SGPT, CPK (Med. Clin. No. Amer. 48:189, 1964)

Uric acid: ↑ → ↓ (Ann. Surg. 158:952, 1963)

5HT, catecholamines, histamine, vasoactive peptides (Surg. Gyn. Obst. 117:315, 1963)

Acid phosphatase ↑ (J. Surg. Res. 9:339, 1969)

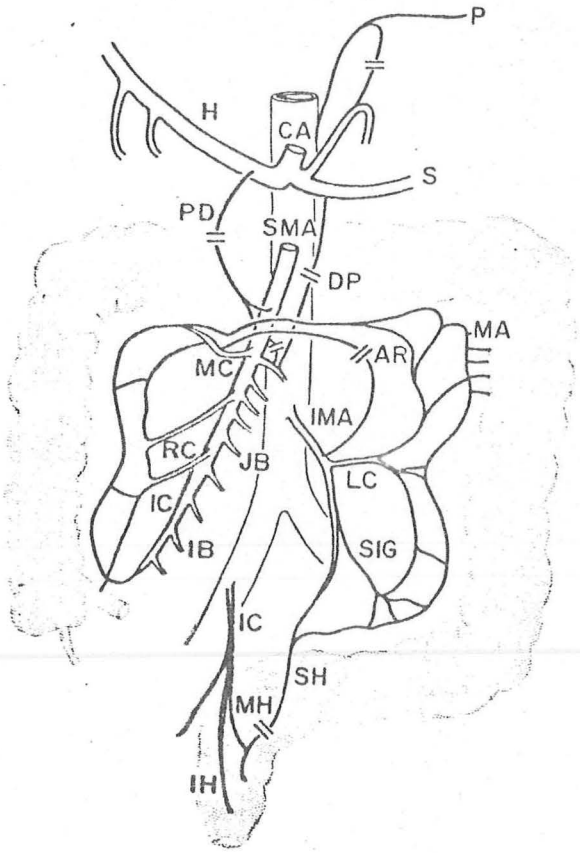
Acute gastric erosions and ulceration: Acute stress ulcers are frequent in patients with ischemic bowel disease. They may cause massive bleeding. It has been shown that in the dog SMA stenosis may increase acid secretion and contribute to ulcerations. However, several observers believe that the so-called stress ulcers may be a manifestation of gastric ischemia. (See Surg. 68:220, 1970.)

Platelet aggregation: It has been shown that patients with intestinal infarction have an exceptionally high incidence of microembolic manifestations in the systemic circulation. These emboli are believed to be "white bodies" composed of platelet aggregates. Platelet aggregation occurs as a result of high levels of circulating 5-HT and ADP and injured mesenteric vascular tree. Vyden has observed platelet "white bodies" in the retinal vessels in patients with mesenteric infarction.

Causes and Pathogenesis of Mesenteric Ischemia

Anatomy and physiology: Anatomy of mesenteric circulation is outlined below:

ARTERIAL SUPPLY OF GUT
SHOWING POTENTIAL COLLATERALS

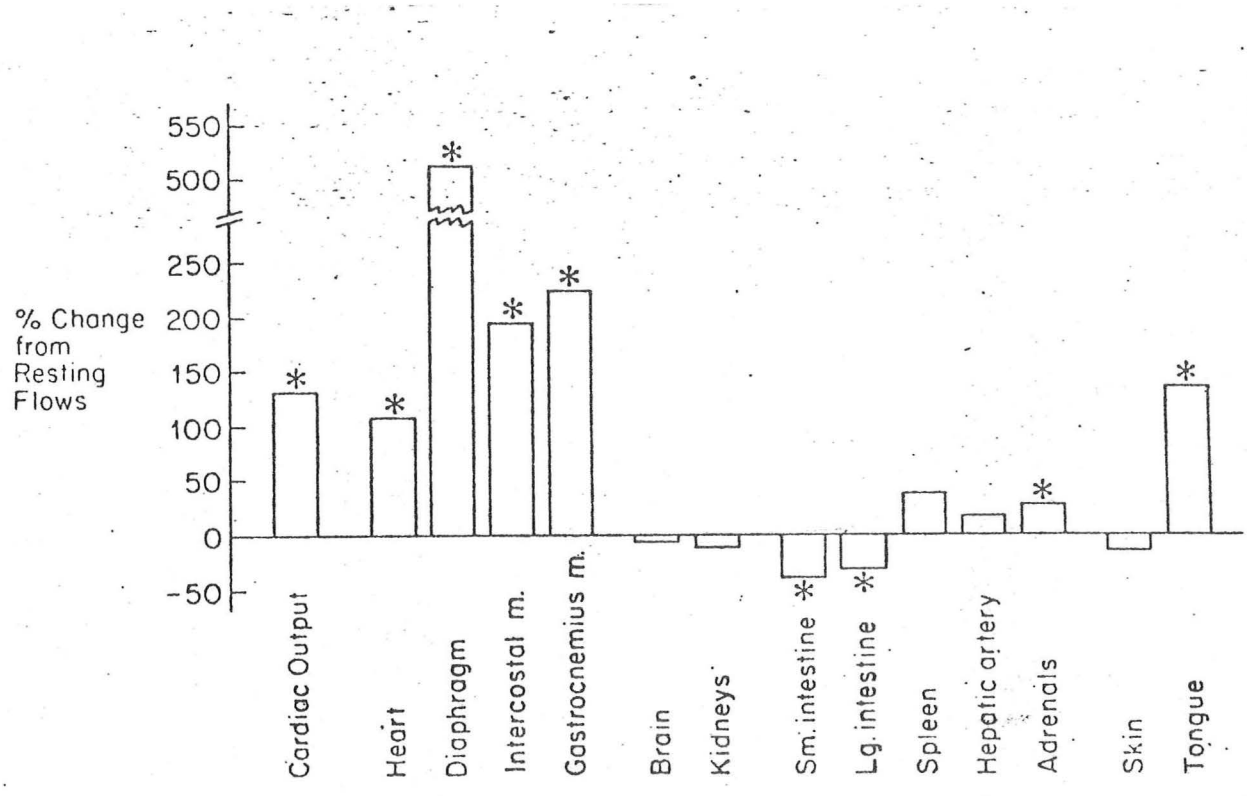


The celiac axis territory which includes liver, stomach, duodenum, pancreas and spleen are richly supplied with blood. Moreover, there are many collaterals between celiac and SMA and between celiac and non-mesenteric arteries. Furthermore, celiac axis may not precipitate in vasoconstrictive response to the same degree as the SMA. As a consequence, ischemia of celiac territory is rare.

SMA supplies the entire small bowel from the ligament of Treitz down and half of the colon. SMA does not form anastomosis with any non-mesenteric vessel, and hence ischemia of its territory is common.

IMA supplies the left half of the colon and makes anastomosis with hemorrhoidal branches of internal iliac. However, in spite of the fact that the colon is supplied by both SMA and IMA, ischemic disease of the colon is fairly frequent because of poor blood flow to the colon.

Normally, splanchnic area receives approximately 20% of the cardiac output at rest. Fixler, Atkins, Mitchell and Horwitz have shown that at rest the flow to the small intestine and large intestine in awake dogs is 46 and 68 ml/min/gm of tissue. However, in the exercising dogs, the blood flow to these organs is reduced more than any other tissue. A moderate exercise of running on treadmill at 6-8 mph for 3-4 min caused about 40% reduction in the blood flow to large and small intestines, as shown below:



(Fixler et al., 1975)

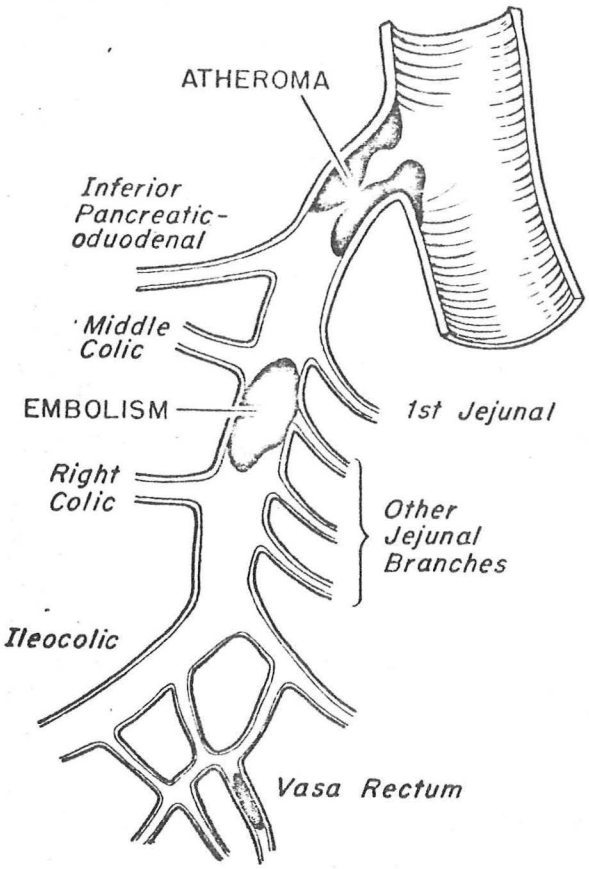
Pathogenesis of mesenteric ischemia:

A rational treatment of ischemic bowel disease depends upon the proper recognition of the cause of ischemia. Moreover, the clinical manifestations produced by different causes vary somewhat and may aid in proper diagnosis. In general, bowel ischemia may be due to: 1) occlusion of the main vessel, 2) occlusion of smaller vessels, and 3) nonocclusive ischemia.

1) Occlusion of main vessels: Generally speaking, the patients with occlusions of main trunk are potential candidates for surgical correction. Both arterial and venous occlusions can cause ischemia. The causes of arterial occlusion in order of frequency are: atherosclerosis, thrombosis, embolism, celiac compression syndrome ??; miscellaneous causes such as: aneurysm, trauma, surgery, A.V. fistula and fibromuscular hyperplasia.

Atherosclerosis: This is the commonest cause of mesenteric vascular disease. Superimposed thrombosis frequently precipitates acute intestinal ischemia. It may also be the cause of chronic ischemia and abdominal angina. The atherosclerotic narrowing is usually present at the opening, and the narrowest lumen is present within 1.5 cm of the aortic orifice. The atherosclerosis tend(s) to be less severe towards the periphery.

OCCLUSION OF S.M.A.



Reiner, Jimenez and Rodriquez in a postmortem study of 88 adults of all age groups found mesenteric atherosclerosis in 68 out of 88 adults. In 49, there was stenosis, and 15 had occlusion of one or more main arteries, 6 had occlusion of SMA, 4 of these had intestinal infarction, and the other 2 had no evidence of bowel infarction. Other observers have reported cases with occlusion of all the three main vessels and with no bowel infarction due to collateral circulation developed with other vessels.

Derrick et al. found 12-86% narrowing of cross-section area of SMA in 33% unselected adult autopsies. In many patients reduction to about half the cross-sectioned area was not associated with ischemic symptoms, and in most of these patients no anastomotic circulation was demonstrated. Celiac axis was narrowed in 44%. Dick et al. (1967) using lateral lumbar arteriography noted that IMA was not visualized in 40% of cases.

Dick et al. found that relative diameter of celiac: SMA; IMA was 4:4.5:1, respectively, which corresponded to cross-section area of 64:81:4, respectively. They found that symptoms of bowel ischemia was not found in patients whose cross-section area of the arteries was greater than 2/3 of the normal. Moreover, 20% of patients with any evidence of ischemia had cross-section area of 2/3-1/3 of normal.

Atherosclerosis may also lead to aneurysm of abdominal aorta and dissecting aneurysms. These can cause occlusion of mesenteric vessels.

Thrombosis: Thrombosis almost always occurs in the presence of marked atherosclerosis and helps to produce complete occlusion of vessels. Very rarely thrombosis of normal vessels can occur. Thrombotic disorders which may predispose to vascular occlusion include polycythemia, thrombocythemia, sickle cell disease, polyartritis nodosa, cryoglobulinemia, thrombotic thrombocytopenic purpura and amyloidosis.

Embolism: They arise from 1) intramural thrombus secondary to myocardial infarction, 2) atrial clot in rheumatic vulvular disease, 3) atheromatous plagues from aortic wall. Rarely, they have arisen from 4) bronchogenic carcinoma, 5) myxoma and 6) aortic vulvular prostheses.

Celiac axis and IMA emboli are very rare. IMA may be involved in saddle emboli to aorta. SMA emboli represent approximately 5% of all peripheral emboli. Most commonly, the embolus lodges at the level of the middle colic artery (55%), but it can occlude SMA (15%), right colic (16%), and ileocolic artery (7%). Distal emboli rarely occur (4%) and multiple emboli have been found.

Venous occlusion: The occlusion of veins is usually due to thrombosis. The thrombosis can be primary, i.e., due to unknown cause or it may be associated with a variety of conditions such as: polycythemia, hypercoagulable states, carcinoma, portal hypertension, sepsis, tumour compression, direct injury and contraceptive pills.

2) Occlusion of smaller vessels: (small vessel disease): A large variety of disorders can involve the small unnamed vessels of the gut. When the involvement is isolated, at one or few small vessels, no ill effects are produced because of rich anastomosis. However, in the presence of generalized small vessel disease, blood flow may be severely reduced. Because in these cases, the ischemia occurs slowly and progressively, and massive infarctions are uncommon. Also, syndrome of intestinal angina is usually not produced. These cases usually produce segmental infarctions, which are generally mucosal but may become transmural.

Various disorders which have produced basal ischemia due to small vessel disease have been outlined below:

Small Vessel Disease

I. *Inflammatory*
   1. Adjacent to an ulcer
   2. Ulcerative colitis and Crohn's disease
   3. Infective angiitis—typhoid, tuberculosis, syphilis, dysentery, leprosy, etc.
   4. Specific arteritis of Crohn's
   5. Radiation
   6. Benign focal arteritis (in appendix)
   7. Buerger's disease

II. *Vasospastic disorders*
   1. Malignant hypertension
   2. Post-coarctation syndrome
   3. Arteriolitis associated with phaeochromocytoma
   4. Vasopressive drugs

III. *Immune complex and collagen disease*
   1. Systemic lupus erythematosus
   2. Polyarteritis nodosa
   3. Rheumatoid arthritis
   4. Dermatomyositis
   5. Sjögren's
   6. Wegener's granuloma
   7. Anaphylactoid purpura (Henoch-Schönlein purpura)
   8. Progressive systemic sclerosis

IV. *Degenerative*
   Atherosclerosis—diabetes mellitus

V. *Miscellaneous*
   1. Hereditary haemorrhagic telangiectasia (Osler-Weber-Rondu)
   2. Amyloidosis
   3. Pseudoxanthoma elasticum (Grönblad-Strandberg syndrome)
   4. Ehler's Danlos
   5. Malignant atrophic papulosis (Köhlmeier Degos disease)
   6. Fabry's syndrome (angiokeratoma corporis diffusum)
   7. Drugs—potassium
   8. Moschcowitz's disease (thrombotic thrombocytopenic purpura; thrombotic microangiopathy)

(Kumar, 1972)

3) Non-occlusive mesenteric ischemia: One of the major advances in our knowledge of the ischemic bowel disease has been the appreciation that a large majority of cases of ischemic bowel disease do not have any evidence of organic vascular occlusion. This is due to the unique ability of the mesenteric circulation to modulate blood flow through it.

In situations in which systemic blood pressure and/or cardiac output are threatened, there may be marked reduction in mesenteric blood flow. When hypotension becomes marked, the mesenteric flow may be reduced to the point of almost complete cessation, at least for brief periods ot time. This occurs in an effort to maintain adequate circulation to the brain and heart. Gut, of course, can and usually does take it -- this may be why it is called the gut. However, this can be overdone with severe consequences. Severe and prolonged vasoconstriction in the face of already compromised mesenteric circulation due to atherosclerosis or small vessel disease may lead to pathologic changes of ischemia in the gut.

Some of the clinical states associated with non-occlusive mesenteric ischemia are outlined below:

Hypotensive shock: As the normal level of blood pressure is threatened, the mesenteric vessels undergo vasoconstriction and the blood flow to the mesenteric system is reduced. The blood flow is further reduced when hypotension sets in. Vatner has recently reported that in conscious dogs blood loss of 14 ml/kg did not cause a significant change in the mean arterial blood pressure (although it caused an increase in heart beat of 17 beats/min) but caused about 20% reduction in mesenteric blood flow. Blood loss of 26 ml/kg caused a drop of 23% in the mean arterial blood pressure, but caused 44% reduction in mesenteric blood flow. Lillehei et al. demonstrated that prolonged hemorrhagic shock (4-5 hrs) leads to infarction of bowel mucosa. These authors also demonstrated that endotoxic shock also caused marked mesenteric vasoconstriction due to alpha-adrenergic stimulation. Intestinal infarction associated with hemorrhagic shock in man has been reported.

Organic heart disease: Cardiac failure results in diminished cardiac output. This causes mesenteric vasoconstriction via reflex sympathetic stimulation. During exercise in patients with CHF, mesenteric flow may be reduced to 40% of the normal.

It has been shown that in the dog induced paroxysmal atrial tachycardia may cause 30% reduction in blood flow and ventricular tachycardia may cause 90% reduction in mesenteric blood flow. Cardiac arrhythmias, particularly atrial fibrillation, can cause a significant fall in cardiac output without there being obvious congestive failure.

Vasoactive agents: Alpha-adrenergic stimulants cause marked splanchnic vasoconstriction. Use of these agents in shock states may reduce the mesenteric flow to dangerously low levels and mesenteric ischemia may result.

Dopamine like norepinephrine constricts the musculoskeletal arterioles, but unlike norepinephrine it does not cause renal and mesenteric vasoconstriction. Dopamine may prove to be useful agent in treatment of shock associated with threatening mesenteric vasoconstriction.

Cardiac glycosides: Several recent studies have shown that splanchnic arterioles are major sites of constriction by cardiac glycosides. Fully digitalizing doses of oubain increase mesenteric vascular resistance in the dog and monkey. Oubain also produces further vasoconstriction in shocked dog. Similarly, Ferrer et al. found that digoxin administered to patients in heart failure intensified pre-existing splanchnic vasoconstriction. Furthermore, in normal conscious human volunteers, oubain in the doses of 0.25 mg induces splanchnic blood flow by 30-40%. It is of some interest to note that Pierce and Brochenbrough found that nearly all patients with non-occlusive mesenteric ischemia were receiving digitalis at the time of catastrophic event. These studies would suggest: 1) Digitalis should not be used as a prophylactic measure in cardiogenic or hemorrhagic shock, as advocated by some observers, particularly if there is any question about mesenteric ischemia. 2) In patients with obvious congestive failure, and mesenteric ischemia, digitalis should be used with care to obtain optimal digitalis effect without producing overdosage.

Other drugs: Diuretics: It has been shown that diuretic agent furosemide (Lasix) lowers peripheral vascular resistance with a resultant increase in limb flow. This phenomenon in conjunction with mesenteric vasoconstriction by digitalis may represent a potential "steal syndrome" which could critically reduce mesenteric blood flow.

Clinical Syndromes of Ischemia of the Gut

Gastrointestinal ischemia produces a variety of distinct syndromes which are either distinct in their clinical features or distinct because they require therapeutic interventions which are somewhat different from the rest of the cases. The ischemic syndrome of the gut can be described in a variety of ways:

1) On the basis of regional involvement: for example, a) segmental ischemia-like gastric erosions, hemorrhagic duodenitis, ischemic small bowel disease, ischemic colitis, ischemic proctitis, or b) massive bowel ischemia.

2) On the basis of depth of involvement of segment of bowel: for example, mucosal ischemia, submucosal hemorrhage or transmural infarction or gangrene.

3) On the basis of acuteness of ischemic process: for example, acute subacute and chronic ischemic syndromes.

4) On the basis of the pathology of ischemic injury; for example, ischemic erosions, ischemic ulcerations, "ischemic colitis" and ischemic stricture.

5) On the basis of etiology of ischemia: for example, main trunk occlusion, small vessel disease, vasospastic ischemia due to low perfusion states (non-occlusive vascular ischemic syndromes) on the type of of vessel occlusion: for example, artery or vein.

6) From a very pragmatic point of view of management they can be divided into surgical (for example, main vessel occlusions, gangrene strictures) or medical types.

Obviously, all of the above and several other considerations apply to each case of mesenteric ischemia. The clinical presentation of mesenteric ischemia is as varied as the various permutations and combinations of the factors will produce, which are truly enormous in number.

Therefore, I have chosen to make some general comments about the presentation and diagnosis of the ischemic bowel syndrome and then to describe some clinical cases which may represent very important clinical syndromes deserving your special attention.

At the PMH, during the last 4 years, there were 76 cases of ischemic infarction of the gut which were proven by pathology. Nineteen out of these were due to strangulation caused by volvulus or hernia. The remaining 57 cases were due to primary vascular disorders. These selected cases represent an unknown fraction of the total cases of ischemic bowel disease seen at PMH. My rough estimate is that there are perhaps 10 times as many cases in which clinically important bowel ischemia occurs.

1) Distribution of ischemic lesion: In these 57 cases, small bowel was involved in 30 (52%), large bowel in 11 (19%), and both large and small bowel in 16 (28%) of the cases.

2) Age and sex: Ischemic bowel disease is usually considered to be a disease of the elderly persons over the age of 50 years. However, in the PMH series, 25 (45%) patients were below 50 years. Age and sex distribution of these cases is shown in Table below.

Age and Sex Distribution of 57 Cases

Age Group	Female	Male	Both
11 - 20	1	2	3
21 - 30	0	2	2
31 - 40	3	2	5
41 - 50	9	6	15
51 - 60	6	3	9
61 - 70	7	7	14
71 - 80	3	2	5
81 - 90	3	1	4
Total	32	25	57

3) Clinical presentation: It should be emphasized at the very outset that the diagnosis of ischemic bowel disease is largely clinical. Awareness and appreciation of this disease is the key to early and proper diagnosis.

The symptom complex of bowel ischemia consists of abdominal pain, diarrhea, bleeding, nausea and vomiting. Abdominal pain out of proportion to the physical findings in the abdomen is the hallmark of ischemic bowel disease.

Pain may not be a prominant feature in some cases. Some autopsy studies note pain may not be prominent in 50% of the cases, but pain is absent only in a minority of clinically recognized cases (0-25%). The pain is usually diffuse and crampy. Diarrhea is usually not prominent and occurs in about one-third of the cases. Nausea and vomiting are also not prominent but usually occur in acute syndromes.

Bloody diarrhea may occur, but usually the amount of blood is small. Evidence of bleeding occurs in about 50% of the cases. In a small number of acute cases, hematemesis may occur. Bleeding, however, is never massive in these

patients.

Abdominal examination usually reveals mildly distended abdomen (18% early and 56-80% later in disease). Abdomen is usually tender, but no rebound may be present. Sepsis of severe peritonitis are reported in 12-38% of the cases. Bowel sounds are increased in early disease, but later they are diminished or may be absent.

When the volume of the ischemic bowel is large, dehydration is frequent and severe, and shock eventually occurs (over 60%). Other systemic effects such as acute myocardial ischemia may be detected. Cardiovascular lesions which may provide basis for the etiology of bowel ischemia are present in a majority of the cases

4) Laboratory findings: are usually not helpful. Hemoconcentration occurs due to volume deficits. Leukocytosis frequently occurs due to dehydration, ischemia and sepsis. BUN is usually elevated. Serum enzyme changes are not consistent to be helpful in diagnosis. In acute state, marked acidosis may be prominent and may be very helpful in diagnosis. Brooks and Carey found that patients with acute mesenteric occlusions have severe base deficits which are not seen in other causes of acute abdomen (see Table below).

Mean Blood Pressure, pH and Base Deficits in Acute

Mesenteric Occlusion and Other Acute Abdominal Conditions

	Mean Blood Pressure	Mean pH	Mean Base Deficit
Acute mesenteric occlusion (7)	114/76	7.27	-15
Acute abdomen, other causes (21)	112/60	7.46	-0.3

Brooks and Carey, 1973

Low albumin and low gamma globulin may occur due to state of protein-losing enteropathy. In chronic cases, tests for absorption may be abnormal.

5) Plain x-ray abdomen: Several reports have described abnormalities on the abdominal films in these patients. But only few reports have quantitated the abnormalities in terms of their frequence of occurrence. Wittenberg and Tomchik found that changes sufficient to make a probable or definite diagnosis were present only in about 21% of the cases. The various changes and the frequency of occurrence have been summarized in the Table on next page. In chronic intestinal ischemia, there are no abnormalities on plain x-ray abdomen.

Frequency of X-Ray Abnormalities on the Plain Film of Abdomen in 62 Unselected Cases of Proven Intestinal Infarction

Radiographic Changes	No. of Cases	%
(A) Probable or definite infarction: Specific changes:	13	21%
- Gas in bowel wall and/or portal system	3	6%
- Thickened bowel wall with changes in mucosal contour	11	18%
(B) Possible Infarction: Some Non-Specific Changes:	6	10%
- Gasless abdomen	3	6%
- Small bowel pseudo-obstruction pattern	23	37%
- Dilatation of small bowel + trans colon	12	19%
- Splenic flexure cutoff	10	17%
(C) Non-Diagnostic	43	69%
Total Cases	62	100%

Wittenberg and Tomchik, 1971

6) Barium studies: In chronic ischemia, there are no specific changes. Valvulae conniventes may be reduced in number.

In acute ischemia, small bowel series will more clearly show the pathologic abnormalities in the bowel.

Edematous, thick-walled bowel loops, which are separated, have acute hairpin turns and appear rigid on serial films. The transit is slowed. The valvulae conniventes may be thickened and reduced in number. The lumen of the bowel may be difficult to fill, and the outer margin of the bowel may present a picket fence or stack of coins appearance. Large submucosal hemorrages may produce thumbprints or may resemble pseudopolyps. Later in the course ulceration and stricture may be detected.

Barium studies should be done whenever indicated. Most observers would agree that there is no real danger of perforation with barium studies. Barium studies should not be done if immediate surgery is indicated or when

angiography is planned for acute massive bowel ischemia. In the ischemic bowel barium stays for long periods of times because of decreased motility.

7) Angiography in ischemic bowel disease: Angiography is the only way to define and to estimate the degree of obstruction and the nature of the obstructive vascular lesions. It should be done in: 1) suspected embolism, 2) suspected chronic intestinal angina syndrome, and 3) acute SMA ischemia.

Visceral angiography in acute segmental bowel disease including ischemic colon disease is not of established value, and should not be done, in my opinion.

Most observers perform midstream aortography before selective catheterization. Most centres stress the importance of prior lateral aortography. This should always be followed by selective catheterization on the major vessels to define the anastomotic channels and the direction of the flow.

8) Careful examination at surgery: If ischemic bowel disease is diagnosed at surgery, obviously gangrenous bowel should be resected, but extensive resections of bowel of questionable viability should not be done. In that case, careful search for any correctable vascular lesion should be made and appropriately corrected. The abdomen is closed and a "second look" operation is planned 12-24 hrs later, so that the resection of clearly dead bowel is performed.

Specific Clinical Syndromes:

Chronic Occlusion of Mesenteric Vessels (Intestinal Angina Syndrome):

Case 1: [redacted] 45 M

[redacted] 73: C: Epigastric pain, ↑ eating; episodes of watery diarrhea; N & V; wt. loss (20 lbs.)

PE: Abd soft; tender LLQ; no rebound; B.S. normal

LAB: WBC = 16,000; UGI = duod deformity

Tt: Antacids, ? relief

[redacted]. 74: CC: ↑ abd pain - 5 days

LAB: WBC = 17,500; left shift; KUB = dilated bowel loops in RLQ;

Dx: Diverticulitis

LAP: Gangrene mid-jejunum; 4 feet; Occl. SMA 1 cm from origin; thrombosis in jejunal branch which supplied the necrotic bowel loop; resection + anastomosis.

Arteriography: Obst to SMA, distal to take off of middle colic; collaterals, gastroduodenal, meandering artery and Drummond's artery

Case 2: [redacted] ([redacted]) 51 F

[redacted]. 73
C: Epigastric pain, ↑ meals; radiation to back, at night; diarrhea - 1 yr
PE: BP 220/120 (Ismelin + Esidrex)
LAB: EKG = sinus brady + LVH & strain;

[redacted] 73
CC: Pain + wt loss (∿ 30 lbs)
LAB: Fecal fat = 20 gm/day
Dx: Pancreatic disease

[redacted] 74
C: Persistent pain, severe, all night, worse in supine position
LAB: Alk phos ↑; WBC = 17,000

[redacted] 74
C: Abdominal pain; wt loss (∿ 60 lbs)
Aortogram: Occ. of all 3 vessels at origin + renal artery stenosis; extensive collaterals
Vascular surgery: contemplated; one night BP ↑ (185/120); given 40 mg i.v. lasix; In morning: severe abd pain; BS absent
LAP: Intestinal necrosis from stomach to rectum

Comments:

1) These cases illustrate the difficulty in making a prompt diagnosis of the syndrome of intestinal angina. There are no specific symptoms. Classical history of pain occurring 15-30 minutes after meals may not be clearcut, or it may be confused with peptic ulcer disease as in Case 1. On the other hand, abdominal pain with radiation to back and weight loss may suggest carcinoma, particularly pancreatic cancer (as in Case 2). Physical examination is usually unrewarding. A high proportion of patients who prove to have ischemic gut disease have bruits, but the reverse is not true. Stools may be guaiac-positive, but are usually negative. Plain x-ray abdomen, BE, upper GI and cholecystography are usually normal, but small bowel may show nonspecific changes consistent with malabsorption. Fecal fat excretion, D-xylose, Schilling test may be suggestive of malabsorption. Biopsy of the proximal small bowel is usually normal or show nonspecific abnormalities. In order to diagnose these cases early, a diagnosis of mesenteric ischemia should be considered in differential diagnosis of obscure abdominal pain, particularly if it is associated with weight loss..

2) Why is recognition of this syndrome important? The proper recognition and diagnosis of this syndrome is important because many of these cases have correctable lesions which can afford good relief, as most of the cases of

intestinal angina syndrome occur due to main trunk occlusion. Second, about 50% of the cases with acute catastrophic ischemic disease give a history of chronic ischemia. Therefore, it is hoped that correction of chronic lesions may help prevent catastrophic and fatal acute episodes, as in Case 2. There is no medical treatment for this condition.

3) The only real method of recognizing correctable lesions causing chronic intestinal ischemia is angiography, i.e., aortography with lateral and anterio-posterior lumbar arteriograms, and when necessary selective catheterization of main trunks.

Problems in angiographic diagnosis: Majority of the cases with syndrome recognized so far, have atherosclerosis of mesenteric vessels as the cause of ischemia. However, atherosclerosis of mesenteric vessels is common and arteriographic abnormalities in some totally asymptomatic cases can be quite similar to those found in patients with frank ischemia.

Dick et al. found that in their cases of ischemic bowel disease, the total cross-sectioned area of the main trunks was between 2/3 to 1/3 of the normal; but 20% of their patients without intestinal ischemia also had similar reductions. They suggested if the total cross-sectioned area is not reduced by 2/3 of normal or less, the symptoms are unlikely to be due to ischemia

The presence of arterial anastomosis is evidence that significant stenosis of the main trunks is present. However, when well developed, they are likely to produce relief of symptoms, and hence these changes should be seen in the light of clinical presentation

In conclusion, arteriography cannot make a diagnosis of gut ischemia, but it can display the anatomic status of the vessels. Therefore, in patients with chronic ischemic bowel disease, it may help guide the corrective surgical therapy.

4) As pointed out above, chronic intestinal angina syndrome is usually due to atherosclerosis of mesenteric vessels; however, other lesions such as fibromuscular hyperplasia, Takayasu's arteritis,aneurysm of aorta, dissecting aneurysm of aorta, SMA aneurysm, and A-V fistula between SMA and SMV may cause a similar syndrome. Recently an interesting case of methysergide causing chronic intestinal angina has been reported.

5) This syndrome is almost always due to SMA occlusion. It has been suggested that extrinsic compression of celiac artery by the median arcuate ligament of diaphragm may produce such a syndrome. But this is questionable.

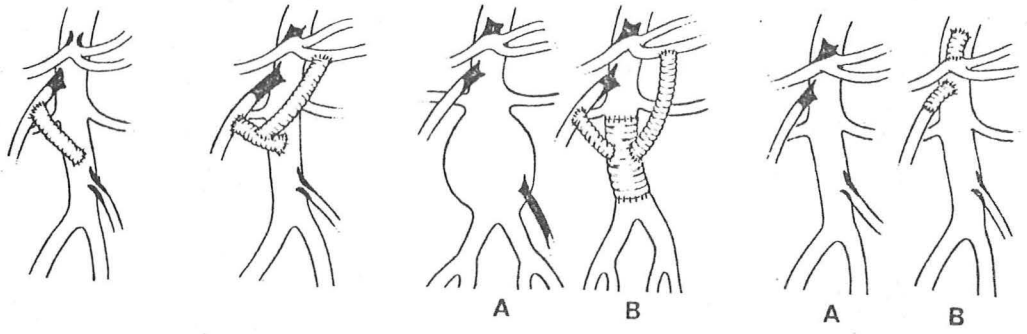
6) Management of these cases: The proper management of these cases is corrective vascular surgery. Correction or bypass of stenosis will relieve symptoms in most cases and may eliminate the fear of acute mesenteric infarction. Many authorities stress the point that once the diagnosis of chronic intestinal ischemia is made and a surgically correctable lesion demonstrated, surgery should be performed as soon as possible.

When the patient has other associated diseases and the overall prognosis is not good, the decision to operate is more difficult, particularly if the symptoms

are not severe. This is particularly so because the natural history of this syndrome is not properly known. For example, although it is known that about 50% of the cases with acute infarction give a chronic history suggestive of ischemia, it is not known as to what per cent of cases with chronic ischemia proceed to die from an acute episode or die of other causes.

A variety of surgical procedures have been employed to relieve obstruction to flow, but bypass operations are most frequently done. Some illustrative examples of these procedures are shown in Figure below. Morris et al. insist that

EXAMPLES OF REPAIR FOR CHRONIC MESENTERIC OCCLUSIONS



proper conditions should achieve the pressure tracing in all the 3 vessels which are identical to the aortic pressure tracing. This is important, because otherwise patient may require second operation before cure is obtained.

In centres with considerable experience in vascular surgery, operative mortality of 10% and postoperative complication rate of around 20% is reported. A review of approximately 100 successful elective revascularization procedures showed that over 90% were relievedof their pain and over 75% improved their malabsorption.

Unfortunately, long-term followup is seldom provided, so we cannot answer the questions: Do these procedures improve life expectancy? Or do they even provide protection from fatal acute mesenteric infarction? Even so, because of the considerable symptomatic relief afforded by these procedures and because of the feeling that it may protect from acute episodes, these procedures should be done if the patient is a reasonable operative candidate.

The medical treatment which is of dubious value is directed against preventing stresses on the compromised mesenteric circulation. Thus, small frequent meals and avoiding exertion after meals may be helpful. Nitroglycerine may be helpful in some patients, but anticholinergics are usually of no avail. Anticoagulants are not considered to be of great usefulness.

Cardiac arrhythmias and potential hypotension and hypovolumia should be recognized and guarded against. Digitalis and diuretics should be used with care and vasopressors should not be used if acute catastrophe is to be avoided.

Syndromes of Acute Massive Bowel Ischemia:

These syndromes involve ischemia of SMA territory with or without ischemia of the other regions. Three distinct types of clinical syndromes under this category are described below:

Case 3: [redacted] 68 M

1971: Hypertensive heart disease with A.F., which failed to respond to cardioversion

Treated with digitalis, diuretics, anticoagulants

[redacted]. 1973: Multiple emboli to different organs; while in the hospital, pt developed acute abdominal pain.

Angiography: Complete occl. of SMA, with no collaterals

At surgery: Pulseless SMA, entire bowel blue; embolectomy performed; second look planned

Second look next day: A small segment of small bowel was necrotic; resected

Did well postop

4 weeks later: Another stroke, cardiac arrest → Died

Case 4: [redacted] 39 F

[redacted] 74: Raynaud's phenomenon left arm; complete occl. of subclavian at its origin; bypass surgery

[redacted] 74: Dorsal sympathectomy

[redacted]rd: (4th postop day), abd. pain, distension and fever; abd. soft, mild tenderness, no BS

[redacted]th: Laparotomy for bowel obstruction; ischemic bowel; 5 feet resected; status of vessels not described

[redacted] 1: Reoperation for anastomosis leak; more gangrenous bowel; most of small bowel (except 1 foot) + rt. half of colon resected; thrombosis of SMA

[redacted] 75: Multiple problems associated with short bowel syndrome

Comments:

Case 3 represents a typical example of SMA occlusion due to embolus and Case 4 is a good example of thrombotic SMA occlusion. As pointed out earlier, for practical purposes SMA should be considered an endartery. Acute occlusion of this vessel leads to extensive bowel ischemia. I would like to make several points regarding these problems:

Frequency: Emboli and thrombosis account for less than one-third of the cases of massive bowel ischemia, but this group is important because it has

potential for proper correction. Emboli and thrombosis probably occur with equal frequency.

Background: Case 3 shows a typical background of cases of embolization. He had cardiac arrhythmia and history of emboli to other organs. The SMA thrombosis almost always occurs in patients with marked atherosclerosis; almost half of the patients may give history of chronic intestinal angina. Our patient (Case 4) did not give history of chronic pain. Many of these patients have evidence of atherosclerotic disease in other organs.

Clinical Features: The clinical features of acute bowel ischemia have been outlined earlier. Thrombotic occlusion may take a more subacute course in development of signs and symptoms as compared to an embolus, but this distinction is not reliable.

Since early diagnosis is the key to good outcome, it is important that we appreciate the early signs of bowel ischemia. As stated before, bowel-wall ischemia proceeds from mucosa to serosa. The diagnosis should be made at the time of mucosal and submucosal ischemia, before transmural infarction occurs. When signs and symptoms of transmural infarction such as generalized or localized tenderness and rebound occurs it is already too late. In an analysis of 71 cases of SMA occlusion, Mavor found that over 70% of the patients had severe abdominal pain for over 12 hrs (median = 12-48 hrs) before they were admitted to the hospital. Moreover, after admission to the hospital, laparotomy was delayed usually for 12-48 hrs.

The only way to make an early diagnosis would be to suspect SMA occlusion on the basis of clinical history plus unimpressive abdominal findings and to obtain early arteriography.

Angiography: If SMA occlusion is suspected, arteriography is the only way to make a diagnosis of vascular occlusion. If occlusion in SMA is found, it could be further differentiated with thrombosis or embolism. In embolism, arteriogram generally shows: 1) occlusion of SMA distal to its origin from the aorta; 2) spasm of artery both proximal and distal to the occlusions; and 3) lack of any collateral flow. In thrombotic occlusion, the aortagram shows: 1) occlusion of SMA, usually at its origin; and 2) slight but inadequate collateral flow.

Management: Proper treatment of SMA embolization is embolectomy. Most authors believe that bowel resection should not be done at the time of embolectomy for two reasons: a) viability of the gut is difficult to determine after a short period of observation; b) bowel anastomosis may break down even if ischemia is controlled.

The patient should be reoperated 6-8 hrs or 12-24 hrs later (according to different observers) for a "second-look" operation. At this time non-viable bowel should be resected and potency of the vascular repair should be assessed. It is important to emphasize that the decision for "second-look" operation should be done at the time of the initial operation, considering the status of the bowel. In the postoperative period, need for second operation for possible gangrene cannot be determined.

Proper treatment of SMA thrombosis is bypass with venous or prosthetic graft or a two-stage procedure. Blind thromboendarterectomy is neither

satisfactory nor safe. Resection of bowel is done at the "second-look" operation.

However, in order that proper management as outlined above can be done, it is important to diagnose these cases early. For, if there is frank necrosis at the time of initial surgery, only hope is bowel resection.

All patients will need massive volume support and antibiotics. Antibiotics should be given because half of these patients have positive blood cultures. Severe volume deficits with hyperkalemia and acidosis may be particular problems in the postoperative period, especially when the revascularization is delayed.

Prognosis: Acute SMA occlusion carries a mortality of over 90%. It is now clear that acute SMA occlusions should be treated as vascular emergency; but, nevertheless, the diagnosis is still commonly made at a late stage of extensive transmural infarction at laparotomy, which in such circumstances is no more than an incident which hastens death. Extensive bowel resection is not an answer to the problem. The patients who survive such resections suffer severe morbidity as gutless cripples.

The mortality rates with vascular correction of obstruction are also high: roughly 30% after embolectomy and 60% in cases with thrombotic obstruction. Those patients who survive and have little or no bowel resection may have malabsorption which tends to improve over a 6-month or so period of time.

Acute Occlusion of Superior Mesenteric Vein:

Illustrative Case:

Case 5: [redacted] 48 F

Crampy abd. pain; distension; with diarrhea; vomiting - 2 days

Abd. tense tender, ↓ B.S.

Clin Dx.: S.B. obstruction

Surg.: Dx: Regional enteritis; resection with re-anastomosis; (venous thrombi were noticed); Specimen: hemorrhagic infarction with venous thrombosis

Postop: Fever + abdominal signs

Reop: Extensive bowel infarction → resected

Autopsy: Mesenteric vein thrombosis

Comments:

Frequency: SMV thrombosis accounts for about 10% of cases of bowel infarction.

Background: About 45% of cases give history of thrombophlebitis. A secondary cause for venous thrombosis is usually not found.

Clinical features: Because these patients have more marked edema of mesentery and small bowel, the following clinical features may occur: pattern of intestinal obstruction; confusion with inflammatory bowel disease (as in this case); abdominal mass is felt in 5-10% cases; GI bleeding is unusual, but bloody fluid in peritoneum is common.

Management: Proper treatment is prompt surgical resection of all of the involved gut and its mesentery. Unlike the other forms of bowel ischemia, involved segment can be determined intraoperatively because the involved mesentery is edematous and its veins exude clots when cut. Direct venous surgery is seldom successful. Incidence of extension of postop venous thrombosis is common, and therefore postop anticoagulation should be done.

Outcome: Mortality rate has dropped from 50-80% to 20%, and recurrence rate is under 20%.

Non-Occlusive Mesenteric Ischemia:

The patients with extensive small bowel infarction, who do not have acute occlusion of SMA are considered in this heading. Many of these patients have partial stenosis of SMA due to atherosclerosis, others have small vessel disease and yet others have no associated organic occlusive disease. Primary vascular surgery is not possible in these patients. Their treatment is largely medical.

Unusual Cases:

Case 6: [redacted] ([redacted]) 65 F

Long standing rheumatoid arthritis

Admitted: Coma, shock (BP = 0/0)

Generalized abd. tenderness and guarding; stools loose; guaiac-negative

Autopsy: Main vessels patent; bowel infarction - coagulation necrosis (not hemorrhagic)

Case 7: [redacted] ([redacted]) 67 M

Admitted: 44% surface area, 3rd degree burn

Pt. developed abd. pain

Tender abd.; rebound; B.S. ↑

Peritoneal tap; malodorous fluid

LAP: Necrotic small bowel; entire ileum and most of jejunum resected; no SMA occlusion

Cardiac arrest; pt. died

Case 8: [redacted] 46 M

Heavy alcoholic; grand mal seizure - previous night

Comatose; BP = 0/0

EKG - vent fib.; recent M.I.

Arterial pH = 7.25; pCO_2 = 26

Autopsy: Bowel from ligament of Treitz to mid-trans colon was ischemic; no occl. of vessels

Comments:

1) One of the purposes of my presentation is to emphasize the frequency of occurrence of non-occlusive intestinal infarction. This appreciation is essential for early diagnosis and proper management of cases of intestinal ischemia. Table shown below summarizes the frequency of non-occlusive bowel infarctions in many series. Roughly, about one-half of the cases are non-occlusive.

Frequency of Non-Occlusive Mesenteric Infarction (NOMI)

Reference	No. of Cases in Series	Cases with NOMI (%)
Glotzer, 1959	31	29%
Blannerhassett, 1960	44	32%
Berger, 1961	110	21%
Marrash, 1962	35	3%
Ottinger, 1967	136	49%
Liavag, 1967	40	32%
Bergan, 1969	22	9%
Britt, 1969	45	36%
Williams, 1973	100	76%
Sharefkin, 1974	48	50%
Previous Reports	611	45%
PMH Experience	57	52%

2) This diagnostic possibility should be considered in patients with cardiovascular disease or hypotension due to any cause who complain of abdominal pain, possibly diarrhea associated with distension and abdominal tenderness. Many

patients give history of experiencing such episodes in the preceding months.

In a collective series of 287 cases of non-occlusive bowel infarction, Renton found that 77% had severe organic heart disease. A third to half of the cases had arrhythmias with or without myocardial infarction. The onset of abdominal symptoms is often precipitated by sudden drop in cardiac output due to infarction, onset of arrhythmias or additional stress of other illness such as pulmonary embolus. A majority of these patients are digitalized, and may have evidence of digitalis overdose.

Approximately 20% of cases of bowel ischemia are due to hypotension not associated with primary cardiac disease. Case No. 7 had hypotension due to burns. Other causes are: pancreatitis, peritonitis, bleeding peptic ulcer, cerebrovascular accident, septicemia and volume deficits.

The temporal relationship between the occurrence of abdominal symptoms and episode of hypotension is variable. Some patients developed abdominal symptoms prior to hypotensive shock. About 25% of all cases may present in this manner (Musa, 1965). In the second group, abdominal symptoms occur after the onset of hypotensive shock. In the third group, shock and abdominal symptoms develop at the same time.

3) There are no characteristic signs and symptoms. In general, the symptoms appear to progress more slowly than those due to occlusive disease. In a collective series, abdominal pain occurred in 55%, vomiting in 29% (with blood in 1.5%), diarrhea in 31% (with blood in 10%).

In a prospective study, Williams et al (1967) reported that out of 7 patients, 6 had abdominal pain early in the disease, another 6 had bloody diarrhea of guaiac-positive stools; a further 4 patients had evidence of peritonitis.

4) Angiograms help to exclude occlusion of major vessels.

5) Management: a) Medical Management: If non-occlusive mesenteric ischemia is diagnosed, surgery should not be undertaken immediately, but vigorous attempts should be made to achieve hemodynamic stability during the 8-12 hrs by: correction of massive volume deficits, control of sepsis, correction of acidosis, improvement in cardiac function, avoid or discontinue drugs which aggravate intestinal vascular spasm such as vasoconstriction and use dopamine to treat shock (alpha-adrenergic blocking drugs plus volume replacement, if feasible).

Local infusion of vasodilators: Recently papaverine and phentolamine have been infused into the SMA through the angiographic catheter. Some claim that it is a very useful procedure.

b) Laparotomy and bowel resection: If non-occlusive bowel ischemia is diagnosed, immediate surgery is not recommended. These patients should have vigorous medical plans to correct the bowel ischemia within 12-24 hrs. At that time exploratory laparotomy is done. (It could be considered as equivalent of "second-look" operation.)

If the diagnosis is first made at laparotomy, obviously gangrenous

bowel should be resected. Extensive resection of questionably damaged bowel should not be done. Some recommend use of splanchnic block with infiltration of perivascular tissue, celiac ganglia and sympathetic nerves with local anesthesia. Abdomen is closed, intensive medical measures started and "second-look" operation done 24 hrs later. Few cases managed in this way have survived.

6) Results: The prognosis of these cases is not good. Only 6 of the 45 proven (proven at exploratory laparotomy) survived. Hard data on the usefulness of therapy in these patients are not available. Most of the data are anecdotal.

If the patient survives the acute episode, good prognosis is anticipated. However, motor dysfunction and/or evidence for mild abdominal sepsis may go on for several weeks, and malabsorption may persist for several months.

Segmental Bowel Ischemia:

Ischemic injury to a small segment of the gastrointestinal tract is much more common than catastrophic massive ischemia. Any segment of the GI tract may be involved. Although transmural ischemic necrosis of stomach and duodenum is very rare, mucosal ischemia producing ulceration and hemorrhagic necrosis are not so uncommon. It has been suggested that the so-called stress ulcers may be due to ischemic injury to the stomach.

Segmental ischemic lesions of the small bowel frequently occur in patients with small vessel disease and those with non-occlusive mesenteric ischemia. They may present as transient mucosal hemorrhage, ulcer, stricture or gangrene.

Segmental ischemic lesions involving the colon are common. They present a wide variety of spectrum. Their recognition is important from the point of diagnosis and treatment.

Case 9: [redacted] - 50 M

Admitted: Valium overdose
In evening: Abd. pain; bloody diarrhea
Slight abd. tenderness, B.S. - normal
BE: Ischemic colitis
OP: For fear of gangrene
Path: Ischemic colitis

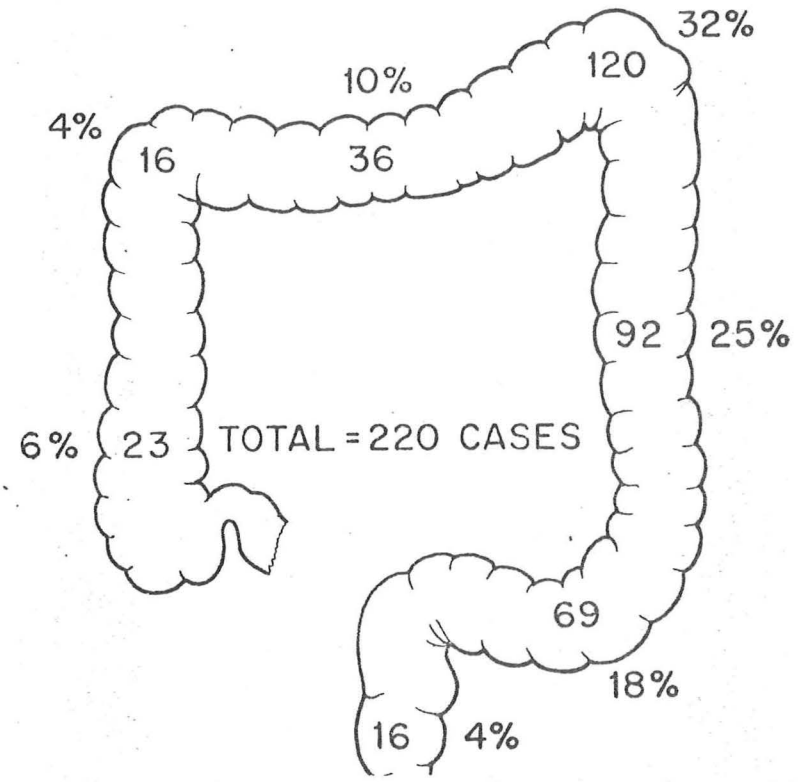
Comments:

Prior to 1950s, cases of isolated colon ischemia were not recognized. In 1950s, increasing number of operations for aortic aneurysm and ligation of IMA resulted in recognition of many instances of colon gangrene and ischemic strictures. Cases such as the one reported above were only appreciated in 1960s. The true incidence of ischemic colon disease is not known, but it appears to be more common

than appreciated. Unfortunately, in the past ischemic nature of many colon lesions was not appreciated, and such cases were described as cases of ulcerative colitis after the age of 50, segmental ulcerative colitis and benign strictures of the colon.

Background: Overwhelming majority of cases of isolated colon ischemia are due to non-occlusive mesenteric ischemia. In a large majority of these cases no obvious cause can be determined.

Clinical features: Distribution of ischemic injury: Distribution of ischemic injury of the colon in a collective series of cases is shown in Figure below. Note left side of the colon is most frequently involved.



The acute ischemic episode is similar to that described earlier, but colon lesions are characterized by the following: a) diarrhea is frequent and bright or dark blood in the stools is common. The bowel sounds are present until late and rectum usually contains blood. Rectal involvement can be diagnosed by proctoscopy and characteristic biopsy picture. Differential diagnosis includes acute infective dysentery, acute diverticulitis, Crohn's disease, ulcerative colitis and carcinoma of colon.

About 10% of cases initially present with transmural infarction. They require immediate surgery.

Barium enema: Early and serial barium enema studies are the key to the diagnosis of colon ischemia. Any elderly patient presenting with abdominal pain and rectal bleeding or bloody diarrhea should have a barium enema within 48 hrs of the acute episode. There is no evidence that carries any increased risk, but it should be done with care. The characteristic finding is thumbprints or pseudo-

tumors. Thumbprints, though characteristic, are not pathognomonic. Therefore, a change in appearance is necessary to make a diagnosis. The submucosal hemorrhages are either resorbed or evacuated over the overlying mucosa ulcerates. Therefore, barium enema repeated one week after the initial studies is either returned to normal or the thumbprint pattern is replaced by segmental colitis pattern.

Important corollaries are: 1) Persistence of the filling defect pattern on repeated studies virtually excludes the diagnosis of ischemic injury. 2) Diagnosis of ischemic bowel disease cannot be made on a single evaluation. 3) If initial studies are delayed, BE may be entirely normal or show a segmental colitis pattern, and hence diagnosis of ischemic nature of the lesion cannot be made with certainty.

In subacute cases, the radiographic appearances may be indistinguishable from ulcerative colitis or Crohn's disease. In others, typical ischemic stricture may form.

Role of angiography: Aortograms in these patients do not usually show occlusion at the origin of either SMA or IMA. Selective angiography of both SMA and IMA has been recommended by some. It has been shown that this investigation may be safely performed in the early stage of the disease. However, majority of the patients show good filling of the marginal artery, suggesting that small vessel disease or perfusion problems are the cause of the disease. Williams argues, however, that if a block of major trunks is found, the patient should have corrective surgery.

Angiography demonstration of occlusions or stenosis of IMA are of no value in the diagnosis.

Course of Ischemic Bowel Disease: There are four possible outcomes of acute colon ischemia:

1) Progression to Gangrene: A minority of cases who start with abdominal pain and rectal bleeding of a few days duration subsequently develop frank peritonitis. This complication occurs in less than 5% of cases.

It is extremely important that these cases be observed carefully and kept properly hydrated. Digitalis, diuretics, fluid restrictions (e.g., for various investigations) may convert partial ischemia into complete acute ischemia and gangrene. This complication should be avoidable. However, if it occurs, it should be recognized and appropriately treated.

2) Complete Rapid Recovery: Transient Colon Ischemia: In relatively milder cases of colon ischemia with submucosal hemorrhage, the blood is resorbed and complete clinical and roentenologic healing occurs in 1-2 weeks. In these patients, symptoms and signs usually subside in 24-48 hrs. The recognition of these episodes may be totally missed by proper barium enema study if not performed in time. In Boley's series, majority of cases of colon ischemia fall in this group. To make this diagnosis, BE should be repeated in 2-3 weeks.

3) Ischemic Colitis: Persistent Ischemic Colitis: In moderate ischemia there is necrosis of the mucosa leading to ulcerations and inflammation. At this stage, ischemic lesion may be quite indistinguishable from ulcerative

colitis or Crohn's disease. However, the inflamed and necrotic areas slough, and this leads to complete healing in 1-6 months, as illustrated by case described by Boley and Schwartz (Ref. 121).

4) Ischemic stricture: Following case is a good example:

Case 10: [redacted] 55 M

[redacted] 74:	Alcoholic; acute pancreatitis
	Crampy lower abd. pain; bleeding per rectum - 1 day
	Procto: Colitis
BE:	Ischemic colitis
[redacted] 74:	Crampy abd. pain; bloating
BE:	Stricture
LAP:	Resection, 8 cm sigmoid colon

Management

Primary surgery of vascular obstruction is not needed, because majority of the patients are of non-occlusive variety. Surgery is undertaken only if there is definite progression, suggesting gangrene or perforation. Under these circumstances, resection rather than colostomy is the procedure of choice. Prophylactic colon resection (as was done in Case 9) is not indicated because recurrence of ischemia occurs in less than 15%. Severe hemorrhage to justify surgery does not occur. When stricture forms and produces obstructive symptoms as in Case 10, surgical resection has to be performed.

Ischemic Injury Associated with Other Colon Diseases: "Colitis" Complicating Carcinoma of Colon: "Colitis" developing de novo in a patient with carcinoma of the colon has been recognized for over 20 years, but it is a relatively rare occurrence (in contradistinction to occurrence of carcinoma in patients with ulcerative colitis). While this form of "colitis" has been recognized as having characteristics different from ulcerative colitis or granulomatous colitis, the exact nature has been speculative, and only recently has this lesion been recognized as being ischemic in origin.

Less than 40 cases of this association has been reported. The "colitis" associated with carcinoma shows a full spectrum of colon ischemia from reversible lesion-stricture-gangrene. Most of these cases occur proximal to carcinoma. The ischemic lesion should be resected along with the carcinoma.

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