Primary Mesenteric Venous Thrombosis *

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IN RECENT years the authors have encountered three patients with infarctions of the small bowel caused by spontaneously occurring mesenteric venous thromboses. These venous occlusions were of particular interest, not only because of lack of related mesenteric arterial involvement, but also because the lesions developed in the absence of systemic or intra-abdominal disorders generally recognized as predisposing toward thrombosis. A review of the experience with these three patients, as well as with 34 others reported in the literature since 1950 (Table 1), convinces us that this *primary* † form of mesenteric venous thrombosis deserves more emphasis and consideration as a distinct clinicopathologic entity.

In 1935 it was first emphasized that infarction of the bowel could result from mesenteric venous thromboses without associated arterial occlusion.[5, 17] Since that time, venous occlusive disease has been variously estimated to be responsible for 15 to 45 per cent of all clinically significant mesenteric vascular accidents, carrying with it a mortality of 50 to 80 per cent.[6, 10, 12, 13, 16] In many reported cases, mesenteric venous thromboses apparently evolved as complications of a variety of predisposing clinical disorders. These included diseases associated with: "hypercoagulable states" (e.g., carcinomatosis, polycythemia); local venous congestion and stasis (e.g., hepatic cirrhosis, extrinsic involvement of portal venous radicles by tumor masses or cicatrizing lesions); and direct injury to the mesenteric veins (e.g., accidental or operative trauma, pyelophlebitis secondary to intraperitoneal suppuration, damaging effects of radiation). There have been, however, a number of patients in whom mesenteric venous thromboses developed, *de novo,* in the absence of such predisposing factors. Berry and Bougas,[1] in 1950, were the first to call attention to this important group when they reported 13 cases of "agnogenic mesenteric venous thromboses." They and other authors [10] estimate that 25 to 55 per cent of all occlusions of the mesenteric venous bed are of this "primary" type.

Case Reports

Case 1. A 51-year-old housewife was admitted to the hospital on February 1, 1956, with a history for 18 hours of severe cramping epigastric pain and persistent vomiting. The vomitus, initially clear and colorless, became dark and bloody just prior to admission. Two bowel movements occurring in the same period were formed and normal in color.

This was her third hospital admission. In the first, 14 months previously, she had undergone bilateral saphenous vein strippings following a long history of symptomatic varicose veins complicated by episodes of superficial and deep thrombophlebitis of the lower extremities. In her second admission, 4 months previously, she successfully underwent thrombo-endarterectomies of the distal aorta and both common and external iliac arteries for segmental thrombotic occlusions of those vessels.

On admission, physical examination revealed a

* Submitted for publication April 5, 1964.

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† Term "primary" as applied to this entity was first suggested by Bussey.[2]

TABLE 1. *Cases of Primary Mesenteric Venous Thrombosis Included in this Review*†

Author	No. Cases	No. Patients
Berry and Bougas, 1950[1]	13	12*
Bussey, 1955[2]	4	3*
Duncan, Ferrell and Hansbro, 1952[6]	2	2
Forty, 1957[7]	1	1** (Case 2)
Gillespie, 1961[8]	1	1** (Case 1)
Holsey, 1958[9]	3	2*
Jensen and Smith, 1956[10]	6	6**
Mersheimer, Winfield and Fankhauser, 1953[12]	3	3** (Case 2, 4, 7)
North and Wollenman, 1952[14]	4	3*
Strohl and Lasner, 1950[15]	2	1*
Authors	4	3*
Total	43	37

* Patient suffered a recurrence.
** Cases documented as primary mesenteric venous thromboses.
† Limited to cases reported since 1950.

pale, acutely ill, middle-aged woman. Blood pressure was 100/70, pulse 100 and regular, and temperature was 37.8° C. by rectum. Pertinent positive physical findings were limited to the abdomen. A well-healed midline incisional scar was noted. The abdomen was soft and minimally distended but exquisitely tender in the left upper quadrant. No muscular spasm or rebound tenderness was evident. Bowel sounds were hypoactive. No masses or tenderness were found on rectal examination.

Hemoglobin was 13.5 Gm./100 ml. and the white-cell count was 28,000/mm.^{3}, with 88% polymorphonuclear forms. Urine was normal, serum amylase 50 Somogyi units. A plain x-ray film of the abdomen was normal.

Shortly after admission a nasogastric tube was passed and dark bloody fluid was aspirated from the stomach. Re-examination of the abdomen revealed that abdominal wall spasm and rebound tenderness had developed in the left upper quadrant. A diagnosis of mesenteric vascular occlusion with infarction of the small bowel was considered and laparotomy was performed.

At operation a moderate amount of serosanguineous peritoneal fluid was present. A segment of small bowel, 146 cm. long and 50 cm. from the ligament of Treitz, was markedly edematous, cyanotic and filled with dark bloody fluid. The mesentery of this segment was also strikingly thickened and edematous and when sectioned, thrombi extruded from the cut ends of the mesenteric veins. Arterial circulation, however, appeared intact and patent to inspection and palpation. The involved bowel and its mesentery were resected and an end-to-end anastomosis restored intestinal continuity. Pathologic examination confirmed the operative diagnosis of hemorrhagic infarction of the small bowel secondary to mesenteric venous thromboses.

Postoperatively, heparin anticoagulation therapy was instituted and effectively continued for 11 days. The hospital course was uneventful and the patient was discharged from the hospital on the 12th postoperative day.

Within 36 hours, however, she returned to the hospital after 12 hours of nausea, vomiting, diarrhea and cramping lower abdominal pain. At that time her temperature was 37.8° C., pulse 110 and blood pressure 105/65. The abdomen was soft and not distended but markedly tender in the right lower quadrant. No abdominal wall spasm or rebound tenderness was noted. A diagnosis of recurrent mesenteric vascular occlusion was considered and a laparotomy performed.

At operation a segment of ileum, 40 cm. in length and 20 cm. from the ileocecal valve was found infarcted. Gross appearance of the involved bowel and its mesentery was similar to that seen at operation 2 weeks previously. Mesenteric venous thromboses in the absence of arterial involvement were demonstrated again. A bowel resection and an end-to-end anastomosis was accomplished. Pathologic examination confirmed the operative diagnosis of recurrent mesenteric venous thromboses with hemorrhagic infarction of the ileum.

Postoperatively, heparin anticoagulant therapy was given and the patient did well for 7 days. Her course then was complicated by a wound abscess which required incision and drainage. Anticoagulants were discontinued for 6 days and re-instituted with dicoumarol on the 14th postoperative

day. She was discharged from the hospital 21 days later while still taking anticoagulants, which were continued over the next 4 months. She has done well in the 8 years following her last operation without any suggestion of a recurrence of mesenteric venous thromboses. Despite the combined resection of 186 cm. of small bowel her nutritional status has remained satisfactory and she reports having two or three soft formed stools daily.

Case 2. A 67-year-old Vermont farmer was admitted to the hospital on November 20, 1957, complaining of severe upper abdominal pain. Four days prior to admission, following his evening meal, he experienced upper abdominal pain which became more severe and was associated with nausea and vomiting of bile-stained fluid. On the day of admission he had two loose, normal-colored stools.

In the past he had recurrent episodes of jaundice, associated with upper abdominal pain, distress, and intolerance to fatty foods. He also had episodes of thrombophlebitis of the lower extremities for ten years.

On admission the patient was restless and in severe pain. Blood pressure was 145/85, pulse 80 and rectal temperature 37.6° C. No icterus was noted. The abdomen was obese and protuberant but soft and readily examined. Tenderness with voluntary guarding was present in the left upper quadrant, but no spasm or rebound tenderness were found. No masses or organs were palpable. The bowel sounds were hypoactive and rectal examination was negative. No evidence of active venous disease in the lower extremities was found.

Hemoglobin was 17 Gm./100 ml. and the white-cell count was 16,850/mm.^{3} with 97% polymorphonuclear forms. Urine was normal and serum amylase was 55 Somogyi units. A plain x-ray film of the abdomen was normal.

A diagnosis of acute pancreatitis was considered and supportive therapy with nasogastric aspiration and intravenous fluids was instituted. Twenty-four hours later, after apparent improvement, the abdominal pain and tenderness suddenly grew more severe, localizing to the left paraumbilical region. Spasm and rebound tenderness were now evident but were notably less marked than anticipated, considering the severity of the subjective complaints of the patient. Repeat x-ray examination of the abdomen revealed two loops of dilated small bowel in the left upper quadrant. A diagnosis of mesenteric vascular occlusion was considered and laparotomy was performed.

Several liters of serosanguineous fluid were present in the peritoneal cavity. A segment of jejeunum, 44 cm. in length and approximately 40 cm. from the ligament of Treitz, was deeply cyanotic, thickened and rubbery-firm. The adjacent mesentery was similarly swollen. On dividing the mesentery, organized vermiform thrombi extruded from the cut ends of the veins. The arteries were pulsating and patent. Except for cholelithiasis no other abnormalities were noted. Sixty cm. of jejeunum, including the involved bowel and its mesentery, were resected and end-to-end anastomosis performed. Pathologic examination confirmed the operative diagnosis of mesenteric venous thrombosis.

At the conclusion of the procedure heparin anticoagulation was started and was continued for 12 days. On the tenth postoperative day dicoumarol was given to provide long-term anticoagulation. The postoperative period was uneventful until the 15th day when the patient had one bowel movement containing bright red blood. Anticoagulants were discontinued and there was no further evidence of bleeding. Proctoscopy and barium studies of the small and large bowel revealed no abnormalities. The patient was discharged on the 37th hospital day. At the present time, more than 6 years later, he is alive and well with no recurrence. In the interim he underwent cholecystectomy, removal of a common duct stone and T-tube drainage following an episode of obstructive jaundice.

Case 3. A 73-year-old retired college professor was admitted to the hospital on June 5, 1961, complaining of severe, cramping right lower quadrant abdominal pain. Three days prior to admission he had intermittent attacks of mild epigastric discomfort associated with anorexia. Twenty-four hours before admission, after taking a cathartic, he had a normal-appearing bowel movement. Following this, severe cramping abdominal pain rapidly localized in the right lower quadrant. This was accompanied by nausea, malaise, chills and sweats.

The patient had a 21-year history of recurrent attacks of superficial and deep thrombophlebitis of the lower extremities which had left him with a severe postphlebitic syndrome. He had been admitted to the hospital on numerous occasions in the past for treatment of phlebitis, cellulitis and ulcerations and for repeated extensive bilateral strippings of both greater and lesser saphenous venous systems. He also had been examined in the past for an episode of melena which only had revealed extensive diverticulosis of the colon.

On admission he appeared acutely ill. His blood pressure was 150/70, pulse 80 and temperature was 37.4° C. by rectum. Significant physical findings were limited to the abdomen which was slightly distended and markedly tender throughout. Diffuse spasm and rebound tenderness were pres-

ent. Bowel sounds were hypoactive. Rectal examination was unremarkable, with normal-colored stool in the rectum. The lower extremities were edematous, bearing the stigmata of long-standing venous disease without evidence of active phlebitis.

Hemoglobin was 15.3 Gm./100 ml. and the white-cell count was 11,900/mm.^{3} with 70% polymorphonuclear forms. Urine was normal. Flat and upright x-ray studies of the abdomen were normal except for blurring of the psoas shadow on the right side.

Laparotomy was performed. Free serosanguineous fluid was present and a 15-cm. long segment of small bowel which was edematous and cyanotic was found in the right lower quadrant. The condition of the mesentery of this bowel was similar; when sectioned it contained veins which were filled with soft, formed thrombi. The arteries, in contrast, were pulsating and appeared patent. The venous thromboses were observed to extend into the mesentery of the normal-appearing adjacent bowel. This necessitated a resection of 80 cm. of small intestine and its mesentery. An end-to-end anastomosis of the remaining segments of small bowel with normal-appearing venous drainage was then effected. Thrombolysin, 100,000 units, was administered intravenously at the time of operation and again in the first 24 hours following operation to aid in preventing early extension of the venous thrombotic process.

Heparin anticoagulant therapy was also begun immediately after operation and continued for the next 10 days at which time Coumadin therapy was substituted and continued for the next 6 months. The patient's course was uneventful and he was discharged on the 14th hospital day. Since his discharge from the hospital and the subsequent discontinuation of anticoagulant therapy he has continued to do well for 32 months.

Discussion

Clinical Manifestations. The clinical course of acute primary mesenteric venous thrombosis, as observed in the cases reviewed, differed in a number of respects from that usually associated with acute mesenteric arterial occlusions. Initial manifestations in this form of venous occlusion were neither as dramatic nor as devastating. The patients frequently experienced prodromata of ill-defined abdominal discomfort, anorexia and changes in bowel habits—ranging from diarrhea to constipation—for a few days, and even weeks in some instances,[14] prior to the onset of severe symptoms. These, in turn, were present for one or more days before the patient entered the hospital and usually consisted of recurrent abdominal pain and vomiting which became progressively more severe and persistent. The location and description of the pain varied, but were usually consistent with the site of disease in the small bowel. Bouts of hematemesis and melena were uncommon. On admission, severe dehydration and cardiovascular collapse were rarely evident. Fever, when present, was usually low-grade with rectal temperatures ranging between 37.2 to 38.8° C. Initial examination of the abdomen revealed distention, tenderness and hypoactive bowel sounds. Notably absent, even with severe subjective complaints, were spasm or rebound tenderness. Indications of "an acute surgical abdomen" became apparent only later in the course of the illness. This discrepancy between the signs and symptoms, which was evident in two of our patients, was also observed by North and Wollenman.[14] Rectal examination most often contributed little or no positive information. The most consistent, but not diagnostic, laboratory finding was leukocytosis with a preponderance of polymorphonuclear forms. X-ray findings were not specific but the presence of rapidly dilating loops of small bowel has been considered suggestive of vascular embarrassment. Free serosanguineous peritoneal fluid noted in our three patients, as in 78 per cent of such cases, has led some to advocate abdominal paracentesis as a means of diagnosis for mesenteric venous thrombosis.[10, 12]

Pathology. The essential features of the gross pathologic findings in patients in whom actual bowel wall necrosis and perforation have not yet occurred were found in each of our three patients. The wall of the bowel was markedly thickened, edematous and cyanotic and the lumen was filled with dark bloody fluid. The mesentery was strikingly thickened and discolored. On cut sec-

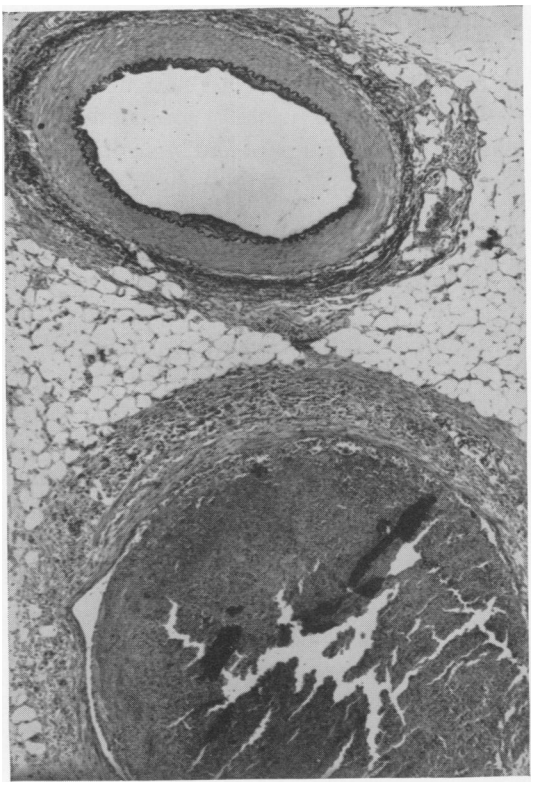

FIG. 1. Photomicrograph of thrombosed mesenteric vein and adjacent normal and patent artery from Case 1. Note absence of inflammatory or fibrinoid degenerative changes in walls of the vessels or surrounding tissue.

tion, thrombi were seen to extrude from mesenteric veins. In contrast, arteries were patent and, if examined *in vivo*, pulsating. Careful examination showed that venous thromboses frequently extended into the mesentery of adjacent, more normal-appearing bowel. This process occurs far more frequently in the superior mesenteric system than in that of the inferior mesenteric. Of 43 cases reviewed in this report there were only two instances in which thromboses involved inferior mesenteric veins.[9, 14]

Microscopic examination of the bowel wall revealed edema, hemorrhage and varying degrees of dissolution of the structural and cellular architecture—changes consistent with hemorrhagic infarction. Of particular interest were the findings in the mesenteric veins. In initial episodes these were filled with bland red thrombi of recent origin which were not extensively fixed to the intimal surface. Adjacent arteries were patent and free of any thrombosis. Veins, arteries and the intervening tissues showed no evidence of inflammatory, allergic or degenerative process that could be incriminated as predisposing to venous thromboses (Fig. 1). However in the one recurrence we observed, fresh thrombi were, in some instances, superimposed on an older organizing process, which could have dated to the previous episode (Fig. 2).

Experimental work of McCune and coworkers [11] and findings in surgical and postmortem specimens suggest that the earliest thrombi originate in veins of the vascular arcades distal to major trunks. From this site thrombi propagate, extending to oc-

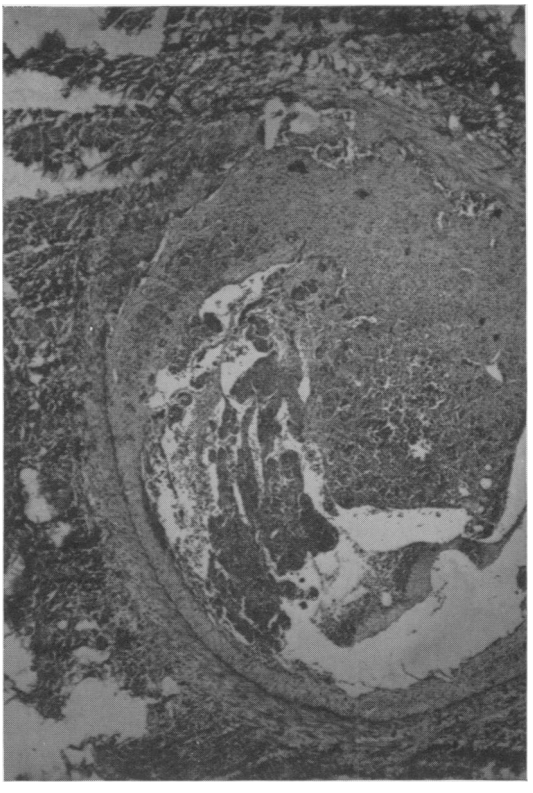

FIG. 2. Photomicrograph of thrombosed mesenteric vein from second operative specimen in Case 1. Note recent thrombotic process superimposed on older organizing process appearing as crescentic intimal thickening.

TABLE 2. *Relation of Treatment to Mortality**

	No. Cases	No. Deaths	% Mortality
Non-operative	5	5	100%
Operative	38	8	21%
Total	43	13	30%

* Data from cases reviewed in Table 1.

TABLE 3. *Influence of Postoperative Anticoagulants on Mortality**

	No. Cases	No. Deaths	% Mortality
No anticoagulants	16	8	50
Anticoagulants	17	0	0
Total	33	8	24

* Only cases in which sufficient data was available for inclusion.

clude collateral channels. A critical point is reached when arcuate, vasa recta and intramural vessels are occluded and hemorrhagic infarction occurs. The main mesenteric channels need not be, and frequently are not, thrombosed at the time infarction occurs. The time interval between onset of the thrombotic process and the final stage when viability of the bowel wall is compromised is largely dependent upon the exact location of the original lesion. If the initial thrombosis is produced in a vein close to the wall of the bowel and extension occurs rapidly, infarction may ensue in a matter of hours. However, if the initial process originates in a larger, more proximally located mesenteric venous radicle and propagation is slow, infarction may not occur for several days or longer. These considerations suggest an explanation for variations in duration of the prodromal stage observed as well as the unusually long duration reported in some cases.

Treatment. Definitive treatment of mesenteric venous thrombosis is surgical. Operation should be carried out as soon as the diagnosis is considered and the patient's condition permits. At operation an adequate excision of the involved bowel and its mesentery should be performed. The surgeon must keep in mind that thromboses frequently extend into the mesenteric veins beyond the limits of the grossly observed infarction. In such instances the resection must include adjacent, apparently normal bowel and mesentery until all venous thromboses are encompassed. If not the hazards of postoperative extension of residual thrombi with subsequent infarction will persist. In most instances if operation is performed early the amount of bowel sacrificed will not compromise the patient's ultimate capacity to maintain nutrition. This was true in Case 1, even though a total of 186 cm., or approximately 50 per cent of the small bowel was finally resected.

In the 43 cases of primary mesenteric venous thromboses reviewed the mortality was 30 per cent (Table 2). Operative treatment was performed in 38 (88%) with a mortality of 21 per cent. In contrast was the 100 per cent fatal outcome of five patients in whom no operation was performed. These operative results compare favorably with those reported for mesenteric arterial occlusions which have an over-all mortality of 88.6 per cent, postoperative mortality of 66.7 per cent and nonoperative treatment mortality of 100 per cent.[10] Thus it would appear that primary mesenteric venous thromboses are more amenable to surgical treatment than are arterial occlusions, but that the former are equally as fatal if not treated aggressively. Also, it would seem that the prospects of surgical success are greater with the primary type of venous disturbance than with that type of involvement of the portal system which is secondary to the effects of other systemic or intra-abdominal diseases.

The role of postoperative anticoagulant therapy in the management of mesenteric venous thromboses has been discussed by others.[3, 10] There has been, however, no agreement as to the value of such treat-

TABLE 4. *History of Thrombophlebitis*

	No. Patients	%
Positive	14	45.2%
Negative	17	54.8%
Total	31*	

* No information available on six patients of the 37 reviewed.

ment. Jensen and Smith [10] concluded that the use of anticoagulants postoperatively did not significantly influence the outcome. Others [3] have suggested that for this reason, plus the additional risks incurred by anticoagulation, such efforts were not warranted. While this may be true when one considers the secondary forms of mesenteric venous thromboses and the morbid circumstances in which they occur, results in cases of primary mesenteric venous thromboses does not support this conclusion. A critical evaluation of the influence of postoperative anticoagulation was possible in 33 cases (Table 3). In the remainder, no information was available as to whether or not anticoagulants were employed. No deaths occurred in cases in which anticoagulants were given postoperatively. This was in sharp contrast to the 50 per cent mortality found in the group in which the drugs were not used. In at least three fatal cases there was postmortem evidence of extension of the original thrombotic process. Thus, despite the relatively small number of cases, postoperative anticoagulant therapy seems to be indicated in the management of primary mesenteric venous thrombosis.

Another critical consideration in the use and duration of therapy with anticoagulants is the recurrent nature of this disease. In this review there were 24 patients treated successfully by operation for initial episodes of mesenteric venous thromboses. Six (Table 1), or 25 per cent of this group, suffered a second bout of this disease. Five recurred within 9 to 40 days and one, 32 months after operation for the first episode. All were successfully managed surgically the second time as well. These experiences suggest that, particularly in the first 6 weeks following the initial episode, etiologic factors originally responsible for mesenteric venous thromboses may persist or recur. If so it would seem that anticoagulant therapy should be continued throughout, and even beyond this period of maximum hazard. Further, lending support to this concept were recurrences in three patients which occurred after short courses of anticoagulants were discontinued. Thus we favor the use of heparin in the immediate postoperative period. After 10 days Dicoumarol or Coumadin therapy is substituted for the remainder of a 6 to 10 week course. One instance of late recurrence, after 32 months, does not seem to constitute sufficient argument for continuing anticoagulants indefinitely.

General Considerations. The etiology of primary mesenteric venous thrombosis is unknown. Venous stasis, intimal damage and hypercoagulability of the blood consequent to a variety of recognized systemic or intra-abdominal disorders have not been implicated. Indeed, the inclusion of a case in this category is dependent on the absence of any such predisposing condition. However, North and Wollenman [14] suggested that thromboses of mesenteric veins may at times represent a visceral manifestation of recurrent migrating thrombophlebitis. In all three of their cases there was an antecedent history of thrombophlebitis. Bussey [2] and Cruikshank [4] also emphasized this possible association which was also true in our three patients and in eight others reviewed. Thus, in 14 of 31 patients with adequate clinical information available a positive history of thrombophlebitis was recorded (Table 4). This incidence of 45 per cent strongly suggests a relationship between the two clinical entities. Furthermore, we feel the process of primary venous thrombosis in the mesentery resembles that

of acute thrombotic processes which occur in peripheral systemic veins. The behavior of the thrombi, the pattern of propagation, absence of direct arterial involvement, recurrent nature, absence of identifiable etiologic features, and pathologic changes are similar. All these considerations lead us to believe that primary mesenteric venous thrombosis may represent a visceral form of the disease which commonly is manifested in veins of the lower extremities. In any case, association of these two disorders should be considered when a patient with a history of recurrent thrombophlebitis of the lower extremities has symptoms and signs of an acute intra-abdominal condition.

Summary

Three patients with primary mesenteric venous thromboses have been presented. The clinical and pathologic features of these and 34 other patients reported in the literature have been discussed. It is believed that this entity should be considered as distinct from arterial occlusions and from more common secondary venous thromboses which develop in the course of a variety of systemic and intra-abdominal disorders. Although the etiology is unknown, we feel that mesenteric venous involvement of the primary type may represent an uncommon manifestation of thrombophlebitic disease more often evident in the lower extremities.

The treatment is adequate excision of the involved bowel and its mesentery followed by administration of anticoagulants. This approach has reduced the mortality previously ascribed to mesenteric venous thromboses. The value of prolonged anticoagulant therapy is also discussed.

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